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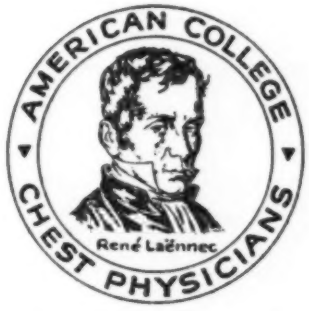

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Fifteen Years' Experience with Carbon Dioxide in the Management of Cough

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Cough is one of the most frequent symptoms for the treatment of which the patient seeks medical attention. The management of a symptom apparently as simple as this is, however, fraught with a number of obstacles. First, often times it is difficult to estimate the usefulness of cough. Secondly, in many instances the potential dangers of certain types of cough are not appreciated and, therefore are left out of consideration. Thirdly, the immediate and incentive causes of cough may not be as obvious as one would be inclined to think at first glance. And finally, a combination of various not readily recognized conditions obviates a priori a stereotyped control of this symptom.

No wonder that the justifiable lament of teachers of medicine, that the management of cough has been more of an art than a science, is being repeated and perpetuated by others. It seems to me that acquiescence is not likely to bring about constructive advances in this field. On the contrary, it seems only as a stamp of approval for using traditional methods of treatment which may not be as competent as they ought to be. It is certain, however, that if the fundamental aspects of this problem are clarified, a great deal can be gained concerning the efficient treatment of cough.

Although cough can be produced voluntarily, it is, in its spontaneous form, a reflex function of the body. Its purpose is the removal of accumulated mucus, inflammatory exudates, products of circulatory stagnation, extravasated blood, or foreign bodies from the respiratory tract and to rid the body from irritation of

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any sort which originates in the air passages. Its most frequent sources are diseases of the lung, the throat and the heart. For the intelligent and efficient treatment of cough it is mandatory to search for its cause and institute appropriate measures accordingly. The multiplicity of lesions which may elicit cough should be recognized. Pathological changes in any part of the respiratory system, whether of infectious, allergic, neoplastic or other etiology, may provoke cough.

The act of coughing consists of three phases, namely the inspiratory, compressive and expulsive. During the first phase an increased amount of air is inspired. At the end of this phase the glottis is closed. During the second phase a forced expiration takes place while the glottis remains closed. In consequence of this forced expiratory effort the chest becomes smaller and the air inhaled during the inspiratory phase is compressed. The consequent pressure of the air contained in the lungs below the closed glottis reaches very high levels. According to Coryllos,[1] it may stand as high as from 80 to 100 mm. of mercury above the normal atmospheric pressure. Other studies revealed as high rise in the intrapulmonary pressure as 160 mm. of mercury. This is equivalent to an increase of about 3 lbs. per square inch above atmospheric pressure. One can readily visualize what a tremendous expulsive force is represented by this high pressure of the air compressed in the lung at the moment the glottis is opened. This initiates the third phase of coughing. The previously compressed air rushes out of the tracheobronchial tubes. Rohrer[2] studied the velocity of air during cough in human beings and found that it varied from ½ to 2½ meters per second in the respiratory bronchioles and from 50 to 120 meters (150-360 feet) per second at the glottis.

According to the concept of Jackson and Jackson,[3] based on their extensive bronchoscopic investigations, the mechanism of cough which results in expectoration consists of two factors: 1, the blast of air ("bechic blast") which is followed by the forcible expulsion of secretions from the larger bronchial tubes; and 2, the tussive squeeze which is a compression of the lung during the expiratory phase of cough. The expiratory contraction of the lung forces the mucopurulent inflammatory products from the alveoli and bronchioles upward into the larger bronchi. Their clear analogy relative to this item is really worth quoting: "For the purpose of illustration, a lobe of a lung may be likened to a sponge partially filled with water. When the sponge is squeezed, the water is forced out."

It is reasonable to assume that these forces of coughing are capable of evacuating inflammatory products or foreign bodies from the respiratory tract. In case of an inflammatory disease

of the lung, such as bronchitis, pneumonia, etc., one would expect a successful drainage under the effect of cough. If this is actually accomplished by coughing, one considers the cough as useful, adequate or beneficial. There is a number of instances, however, when cough is not productive of any sputum or when the amount of sputum expectorated is much less than the coughing effort should have produced. This type of cough is customarily designated as unproductive or useless. Useless or inadequate cough may be brought about by the following circumstances: 1. The source of irritation which initiates the cough reflex is outside of the lung, as for instance in diseases of the paranasal sinuses, an elongated uvula, pressure on the trachea or bronchi by mediastinal inflammation, tumors or dilated blood vessels. 2. Non-inflammatory lesions of the bronchial tubes, such as benign or malignant tumors, may cause persistent, annoying cough without expectoration. 3. During the course of a great many inflammatory involvements of the bronchi or the lung parenchyma there is a phase when the formation of exudate is practically nil, nevertheless the congested state of the involved structures leads to strenuous but useless cough. One of the best examples of this is the first stage of an acute bronchitis with a so-called "dry" cough. 4. A frequent source of inadequate cough is any disease of the lung in which the mucoid or mucopurulent products of inflammation are so tenacious, sticky and adherent to the walls of the respiratory passages that even intense, exhaustive coughing is unable to remove them. The prototype of inadequate cough of this type is that seen in the paroxysmal stage of whooping cough. 5. Accumulation of too much transudation in the alveoli in heart failure may elicit coughing which is insufficient for the cleansing of the lung. 6. There are cases where a retention of pulmonary exudates is taking place because their removal is blocked by pathological structural changes such as the formation of bronchial strictures. Such condition often causes strenuous but ineffective coughing. 7. Atelectasis may be the source of useless cough for the reason that when the lung becomes airless distal to inflamed areas, there is no chance for the inspired air to get behind the inflammatory products which accumulate in the corresponding bronchial tubes and, therefore, it cannot be compressed and act as the normal expulsive force of coughing. 8. Useless cough is common in pulmonary emphysema. It is the result of: a, weakening and destruction of the elastic structures of the lung; b, change in the intrapleural subatmospheric (negative) pressure so that it is near atmospheric (neutral) pressure, and c, the abnormally low (inspiratory) position of the diaphragm. In consequence of these three pathological alterations, the normal phases of coughing cannot be carried through and

cough becomes inefficient, though it is persistent and troublesome. 9. Protracted, unproductive cough may result from fatigue and exhaustion of the expiratory muscles. It is brought about by conditions where there is only slight inflammatory exudation in the lungs but the pathological changes are conducive to coughing. Such is the case in certain types of long-standing tuberculosis, and pulmonary fibrosis.

It is important to realize that oftentimes cough as a reflex mechanism is working with a high degree of failure which may be well designated as *tussic insufficiency*. Cough of this type is unquestionably a liability rather than an asset. Assuming that there is inflammatory exudation in the lungs, its removal either by resorption or by expectoration is essential for the restoration of the normal anatomical and functional status of this organ. In the presence of tussic insufficiency, an accumulation and retention of mucus and purulent exudate takes place. The stagnation of inflammatory products in the bronchi is dangerous: 1, because it may cause complete bronchial obstruction and a consequent atelectasis in the corresponding distal segment of the lung, and 2, the pathogenic microorganisms which are prevalent in the retained mucopus are likely to lead to pathological changes in the surrounding bronchial walls and thus cause the development of bronchiectasis. When massive atelectasis of one lobe or multiple patchy (lobular) atelectasis develops, not only useful respiratory surface area of the lung is lost but also the atelectatic areas must be looked upon as sites of lesser resistance which favor the development of new foci of infection. New areas of disease, of course, are followed by additional production of inflammatory exudate and consequently, by increased cough. A similar vicious circle is bound to result when the bronchial wall is involved by microorganisms in the stagnating mucopus. The force of the compressed air in the lung at the beginning of the expulsive phase of cough exerts a certain degree of pressure upon the bronchial walls weakened and damaged by a severe or chronic inflammation and thus dilates and deforms them. This chain of events is an important etiological item in the pathogenesis of bronchiectasis. The additional increase in cough associated with bronchiectasis thus becomes a serious problem.

Tussic insufficiency may entail other difficulties. Droplets of purulent, thin exudate may be sprayed from one segment of the lung to another or from one lung to the opposite side. When partial bronchial obstruction is present, it may cause a rebound of droplets of infected material and cause their settling in the deeper parts of the lung. In either instance a spread of the disease, which is the original source of the cough, may ensue. It has been recog-

nized for sometime that long-continued cough is liable to produce emphysema. This concept is clearly formulated by Christie.[4] He stated that in chronic bronchitis and in any other pulmonary lesion in which the patient coughs scores of times a day the excessive stress and strain, repeated over many years, causes a destruction of the elasticity of the lung tissue. When pulmonary elasticity is lost, the lung will lack its normal capacity to recoil during expiration. Consequently, the traction or suction effect of the intrapleural subatmospheric (negative) pressure maintains the lung in its inspiratory position. This is a plausible explanation of the typical emphysematous or so-called "barrel-chest". In addition to this, the negative intrapleural pressure dilates the individual alveoli by its pulling effect and thus contributes to the degeneration and rupture of interalveolar septa and to the formation of superficial emphysematous bullae. The greater the dilatation of the lung the closer the intrapleural subatmosphoric pressure will approach the atmospheric level. This, in turn, implies a deficient function of the lung the consequences of which are the persistence and incompetency of coughing in emphysema. It can be seen from this sequence of events that protracted, useless cough is a self-defeating act.

Tussic insufficiency associated with severe coughing interferes with the rest of the patient and with the rest of the lungs. It is a matter of common knowledge that pulmonary rest and relaxation are the cardinal prerequisites of the treatment of tuberculosis. For this reason the harmful effect of inadequate cough in this disease requires no further elucidation. Also, it is known that protracted, hard cough in pulmonary tuberculosis may accelerate the development of specific laryngitis. Pulmonary hemorrhage may be initiated by excessive, strenuous cough. Other possible disagreeable sequelae of severe coughing include spontaneous pneumothorax, vomiting, loss of appetite, exhaustion, headache, insomnia, rise in temperature, marked dyspnea, cyanosis, thoracic pain, fracture of ribs, mediastinal emphysema, subcutaneous emphysema, subconjunctival hemorrhage, urinary incontinence, and, indirectly, myocardial failure.

Reviewing these data and considering the high frequency with which useless and inadequate cough (tussic insufficiency) is encountered in everyday medical practice it is obvious that our efforts must be focused upon the best possible way of its treatment. With this purpose in mind, it is reasonable to say that the efficient management of cough consists of measures which are capable of removing the maximum amount of sputum with the least frequent and most effortless cough.

Reference has been made previously to the muddled state of

affairs which prevails in the treatment of this condition. But how could it be otherwise if the commentary of Clark[5] made only a few years ago is true? He said that unfortunately the pharmacology of cough has scarcely advanced at all in the last half century. Consequently, shot-gun therapy is still popular in this field, and cough mixtures tend to be very complex and completely irrational. Apropos of this, I believe I am not mistaken in stating that such circumstances may explain in a large measure the success of some widely-advertised and euphemistically named pharmaceutical preparations the presumably effective constituents of which are far below the therapeutically useful level.

The correction of this confused situation is attainable only through a more thorough familiarization of ourselves with pertinent physiological facts.

1. The cough center is located in the medulla close to the sensory vagus center and the vomiting center. It is subject to peripheral and central stimuli. It is depressed by alkaloids of opium, alcoholic intoxication and full general anesthesia. With reference to the suppression of cough by some of the opiates, it is well to remember that alleviation of cough in this fashion must not be interpreted as a cure of the underlying disease. Such easy success does not relieve the physician of the obligation of establishing a correct diagnosis.

2. Cough is provoked by the stimulation of the sensory nerve endings of the vagus and glossopharyngeus.

3. The secretory function of the glands of the bronchial mucosa is increased by stimulation of these vagal nerve endings.

4. There is a rich supply of smooth muscles in the wall of the bronchioles. They are arranged in a circular or net-like fashion. It may not be amiss to mention that about one-half of the weight of the lung tissue is made up by these muscles. They have an important role in the normal functioning of the bronchial tubes. Experimental observations revealed that there are a rhythmic systole and diastole of these structures. Spasm of the bronchial muscles is a characteristic feature of bronchial asthma. It occurs also as a result of irritation from other causes, such as the inhalation of irritant gases, fumes, and foreign bodies. Bronchial spasm can easily cause complete occlusion of the smaller branches of the respiratory tract and thereby lead to dyspnea, anoxemia, atelectasis and increased cough.

5. It has been demonstrated with the aid of bronchocinematography that the bronchi and bronchioles have a peristaltic motion directed from the smaller structures toward the larger ones. It is maintained by the smooth muscles, and is independent from the respiratory movement of the lung. The function of this peristaltic

motion is—in analogy to that of the intestinal tract—the evacuation of the bronchial contents. The normal secretion of the mucosal glands as well as inflammatory products or minute dust particles are propelled and expelled by bronchial peristalsis. Drugs of the parasympatheticomimetic group, which cause bronchospasm, also decrease or abolish bronchial peristalsis and thus deprive the lung of one of its natural protective mechanisms. The inhalation of a suitable mixture of carbon dioxide and oxygen is followed by increased peristalsis of the bronchi and the bronchioles. The increased peristalsis is attributable to carbon dioxide.

6. Another factor which is instrumental in the elimination of mucus and foreign particles from the bronchial tract is the function of the cilia. Under normal circumstances ciliary motion is active in the larger as well as in the smaller bronchi. The terminal bronchioles are not provided with cilia. The motion of the cilia is synchronized so that it is capable of driving inert material from the deeper segments of the lung toward the larynx. When a pathological process destroys the sensory nerve endings of the vagus, a cessation of the ciliary function takes place. Also, in areas where the structures of the bronchial wall are lost due to disease, the ciliary action is absent. Such is the case in bronchiectasis. The observations of Negus[6] on the nasal mucosa revealed that the topical application of mildly alkaline solutions of magnesium (pH8 - 8.5) stimulates ciliary motion. On the other hand, acids paralyze it. In view of this, one could conjecture that some of the benefits derived from the administration of alkaline salts in cough may be due to their effect upon the cilia.

7. Gordonoff[7] considers the kinetic force of the respiratory air a contributory factor in removing secretions and products of inflammatory exudation from the alveoli to the smaller bronchi. Partial bronchial obstruction or spasm of the smooth muscles, which is particularly effective in the terminal bronchioles interferes with or eliminates this force entirely from the corresponding area of the lung.

8. Also there exists a force of the secreted bronchial mucus which maintains a slow but continuous motion toward the tracheal bifurcation. Diseases or drugs which exert an unfavorable influence upon the consistency or the amount of bronchial secretions are likely to upset this process. Such may be the case when prescribing morphine as a cough sedative, for it is known that morphine suppresses normal bronchial secretion.

9. A great many experimental and clinical studies concerning the efficacy of various expectorants are open to criticism because their conclusions were based on measuring the amount of bronchial secretions expectorated or otherwise collected. The weakness of

these data lies in the fact that the resorption of some of the bronchial secretion by the bronchial mucosa itself was left out of consideration. Unquestionably, under favorable circumstances a portion of the bronchial secretions is resorbed by the mucosa. Gordonoff[7] pointed out that the scanty, thick secretions of the alveoli are made resorbable either by a process of digestion in the alveoli themselves or by a dilution and liquefaction through the admixture of secretions of the glands of the bronchial mucosa.

10. In addition to the capacity of the bronchial mucosa to resorb exudates and minute foreign particles, a similar process is being carried out in the alveoli. No doubt these two pathways of elimination are of tremendous importance in ridding a diseased segment of the lung of increased and pathological elements. The resorptive capacity of the lung tissue can be effectively increased by certain medicinal measures.

11. The role played by the respiratory motion of the lung in the removal of inflammatory products from the bronchi is not sufficiently appreciated. Under normal circumstances the inspiratory expansion of the chest wall is associated with an increase in the negativity of the intrapleural negative pressure. This, in turn, causes a stretching and dilatation of the bronchial tubes. These rhythmically repeated motions tend to remove the bronchial contents mechanically. Liquid material is thus readily propelled and tenacious, adherent mucopus is separated from the bronchial wall and expelled. This expulsive force is greatly reduced or entirely missing in superficial respiration because the stretching and dilatation of the bronchi are absent. Patients who are debilitated, those under the effect of general anesthesia following major surgery, and some in whom the respiratory center is depressed by narcotics, particularly morphine, may show evidence of insufficient respiratory excursions of the chest. Henderson[8] emphasized the significance of anoxemia in this respect, stating that oxygen deficiency, if at all intense, acts as a sort of whip, which excites respiration to activity and even to excessive activity, but injures at the same time and is liable to be followed by a subsequent period of depressed breathing. The dangers inherent in the retention of inflammatory exudates in the lung (possible atelectasis, bronchopneumonia, bronchiectasis, dyspnea, increased cough) call for immediate measures which are capable of restoring the normal self-cleansing function of the lung. The sovereign remedy of such condition is the inhalation of a mixture of carbon dioxide and oxygen. Its effectiveness is attributable partly to the fact that carbon dioxide is a powerful respiratory stimulant and induces increased inspiratory movements of the thorax which, in turn, cause a stretching and dilatation of the bronchial tubes.

12. It is of more than academic interest to remember that the presence of inflammatory products in the smaller bronchi does not, as a rule, elicit cough irritation, or when it does so, the consequent cough is feeble and inadequate. The fluoroscopic studies of Reinberg[9] revealed that secretions in the peripheral bronchial tree produced no cough in bronchi of the fifth, fourth and third order. Brown and Archibald[10] recorded their observations while operating under local anesthesia upon a patient with a long-standing pulmonary abscess, in the base of which there opened a number of bronchi and bronchioles. They found that cough was readily initiated when a probe was inserted into one of the larger bronchi, while a similar procedure was without effect in the case of bronchioles. Also Jackson[3] observed during bronchoscopic examinations that the finer subdivisions of the tracheo-bronchial tree and the alveoli showed decidedly less cough production from instrumental contact than the larger bronchi. When a pathological process is localized predominantly to the bronchioles, as is the case in acute bronchiolitis, the accumulation of secretions may lead to considerable respiratory distress, anoxemia and cyanosis. In the absence or insufficiency of cough in these cases, therapeutic intervention is imperative for draining the small bronchi and bronchioles and for the relief of the patients' distress. The method applied must not increase the accumulation of exudate. The administration of a mixture of carbon dioxide and oxygen is the measure of choice.

I have been administering carbon dioxide as an expectorant since 1930. Its administration is a simple and safe procedure which does not interfere with the comfort of the patient. In my experience it proved to be a most efficient expectorant. The benefits derived from its use are noticeable subjectively and objectively: (a) spells of strenuous, exhausting coughing are prevented and thereby rest is secured for the patients and particularly for the lungs; (b) an unproductive cough is transformed into a useful one; (c) directly after inhalation the amount of expectorated sputum is increased and its character changes from a heavy, thick and tenacious type into a thinner, serous and more watery kind; (d) the use of expectorant drugs and narcotics can be reduced.

In addition to its previously mentioned pharmacological actions, carbon dioxide is a good expectorant: 1, because it stimulates the myo-elastic structures of the lung and causes a forceful peristaltic movement of the bronchi; and 2, because it liquefies mucopurulent inflammatory exudates that stagnate in the bronchial tract.

Miescher[11] in 1885 called attention to the importance carbon dioxide plays in respiration and formulated the aphorism: "Over

the oxygen supply of the body carbon dioxide spreads its protecting wings." This was followed in 1905 by the experimental work of Haldane and Priestly[12] who actually demonstrated that the carbon dioxide of the blood normally controls respiration. They observed that an increase of the carbon dioxide in the pulmonary alveoli was consistently accompanied by an increase of the respiration. As low as a 1 per cent increase was sufficient to stimulate the respiratory center and to cause deeper breathing. Hill and Flack[13] reported that the inhalation of increasing concentrations of carbon dioxide caused a proportional stimulation of the respiratory center until 10 per cent was reached; higher concentrations led to a progressive depression of respiration. Brown[14] found that the maximum stimulation of pulmonary ventilation was reached at 10.4 per cent carbon dioxide. The greatest protagonist of the clinical application of carbon dioxide was Henderson,[8] who with Haggard and other associates made numerous contributions to the medical literature on the practical value of this method of treatment. They used it for controlling the paroxysmal stage of whooping cough, for the prevention and treatment of postoperative massive atelectasis and pneumonia and for the management of a number of other conditions in which the respiratory center is depressed.

Short inhalations of 5 to 10 per cent carbon dioxide with ample oxygen induce no ill effects. The most conspicuous manifestation of their influence is an increase in the respiratory minute volume. In a self-experiment with 10 per cent carbon dioxide and 90 per cent oxygen, taking 6 liters per minute through a well-fitting face mask my respiratory rate rose from 14 per minute before the inhalation to 19 at the end of a fifteen minute observation period. Henderson,[8] using 5 per cent carbon dioxide, found that the volume of breathing increased more than threefold. Also, marked augmentation in the respiratory minute volume was recorded by Heller and his associates,[15] Barcroft and Margaria,[16], Hitzenberger[17] and Grueneberg and Viethen.[18] The effect usually becomes noticeable during the first minute of inhalation. Brief interruptions render the respiratory center more sensitive. Carbon dioxide causes one to breathe not only more times per minute but also with fuller lungs. It quickens the rate both of the air inhaled and of the air exhaled. There are considerable variations in the same person on different days. Some persons show only slight increase in minute volume to 5 per cent, even to 7 per cent carbon dioxide.

Hartl[19] found that oxygen intake is increased at the beginning of the inhalations, it may remain the same or may decrease during the treatment. Saklad[20] observed that when normal oxygenation was accomplished in anoxemic patients, the respiratory rate di-

minished. The experimental studies of Prinzmetal[21] revealed that the increased respiratory motions of the chest during carbon dioxide inhalations are associated with a greater negativity of the intrapleural pressure on inspiration and a reduced negativity on expiration; both these changes contribute effectively to a competent diastole and systole of the lung. Henderson[8] postulated an increase in the tonus of the respiratory muscles of the chest, particularly the diaphragm, and of the myoelastic structures of the lung during carbon dioxide inhalations. The latter assumption was proved to be correct by the clinical observations of Brunn and Brill.[22] They studied the effect of carbon dioxide during bronchoscopy and noted that it induced violent movements of the bronchial tree and alterations in the shape of its branches: consequently bronchial secretions spilled from minor into major bronchi.

The inhalation of 1 to 8 per cent carbon dioxide improves the functional capacity and the ventricular output of the heart;[17] also it increases both the systolic and the diastolic blood pressure[23] and is followed by an increase in the pulse rate and in the pulse pressure, particularly after the concentration of carbon dioxide has reached 3 per cent.[24] Tomaszewski and his co-workers[25] administered 8 per cent carbon dioxide to healthy persons, and they found, besides the respiratory changes, a slight increase in the metabolism and an initial quickening and then slowing of the pulse. No increase in the metabolic rate was noted by other investigators. Fuchs[26] observed that a slight increase in the blood carbon dioxide accelerated clotting, while too much carbon dioxide in the plasma caused prolonged coagulation. Tannenberg[27] considered this accelerated clotting an indirect transient effect of carbon dioxide on the peripheral and central nervous system that leads to an enlarged production of epinephrine.

I have made observations on 40 patients concerning the respiratory pattern during carbon dioxide inhalation. All of them were given inhalations as a therapeutic measure for the treatment of otherwise uncontrollable cough and dyspnea. The patient was seated on a chair, and the technic and purpose of the procedure was explained to remove fear or apprehension. After the determination of the respiratory rate and pulse rate in this position, from two to five counts were taken during a ten to fifteen minute period of inhalation of the gas mixture. Ten of the 40 patients took the treatment with the aid of a face mask, by the closed method; the others inhaled the gas through a glass tube. In the latter cases the patient was instructed to hold the end of the glass tube in his mouth, to inhale the gas coming from the tank and to exhale through the nose. Although the admixture of air with and dilution

of the inhaled gas was unavoidable, judging from the therapeutic response this method of administration proved to be satisfactory. Altogether 275 observations were analyzed: 17 treatments by the closed method and 258 treatments by the open method (through a glass tube).

By the closed method the amount of gas delivered to the patient varied from 4 to 6 liters per minute. The anticipated response to such high concentrations of carbon dioxide was an increase in the respiratory rate. Still, we found this in 3 instances only. The respiratory rate remained unchanged in 3 and decreased in 9 observations. This is at variance with findings in normal persons and can be explained (1) by the reinflation of previously atelectatic areas of the lung with a consequent immediate relief from dyspnea and (2) by the increase in the amplitude rather than in the rate of respiration. In 2 instances the rate dropped in four minutes from 26 to 14 and from 24 to 10, respectively. In 2 patients an initial decrease in the rate was followed by a moderate increase toward the end of the treatment.

With the open method the pattern of respiratory response was similar to that found with the closed method.

Of the 258 observations with the open method, 4 liters of the gas mixture per minute were given in 8 instances, 4.5 in 9; 5 in 181; 5.5 in 3; 6 in 9; 6.5 in 9 and 7 in 39.

At the end of the inhalations the number of respirations was increased in 89 instances. The increase varied from less than 10 per cent to 60 per cent of the initial rate; the great majority showed a rise less than 30 per cent. In 93 instances the number of respirations was decreased; the reduction varied from less than 10 per cent to 42.6 per cent; it was less than 20 per cent in 79 instances.

The initial respiratory rate varied between 16 and 40; the great majority were between 20 and 29. There was a group of observations in which during the inhalation of carbon dioxide the respiratory rate first decreased and then increased; in another group an opposite response was recorded. In cases in which the rate at the end of the observation period was the same as the original rate, there was an intermediate decrease in 27, or 35.5 per cent, and an intermediate rise in 15, or 19.7 per cent. In the group with a final reduction in the respiratory rate an intermediate increase was noted in 31, or 33.3 per cent. In cases in which the final respiratory rate was elevated, an intermediate decrease was seen in 6, or 6.7 per cent. The intermediate increase varied from less than 10 per cent to 60 per cent of the initial rate, the great majority showing a rise between 10 and 29 per cent. The intermediate decrease varied from less than 10 per cent to 42.6 per cent of the

initial rate, the great majority falling between 6 and 19 per cent.

It is interesting to note that the final respiratory rate was the same as the initial rate in 76 instances, or 29.5 per cent, and it was less than the initial rate in 93, or 36 per cent. Altogether, the final rate remained unchanged or was reduced in 169 instances, or 65.5 per cent, as against 89 instances, or 34.5 per cent, in which the respiratory rate was increased at the end of the inhalation of the gas mixture.

The apparatus used in my work consists of a tank, containing a mixture of 10 per cent carbon dioxide and 90 per cent oxygen or of 5 per cent carbon dioxide and 95 per cent oxygen, mounted on a small platform on casters that makes it possible to give the inhalations to a number of bed patients. Originally, I used an ordinary mask used for general anesthesia; more recently the B. L. B. mask has been found preferable. An oxymeter regulates the flow of gas per minute. The inhaler is connected to the tank by a rubber tubing. A rubber bag which serves as a small reservoir is attached to the inhaler. In some patients it may be expedient to give the inhalations through a glass tube instead of a mask: either because they may be reluctant to accept the mask, or the respiratory stimulation is too strong from the inhalation of 10 per cent carbon dioxide. It is fully realized that when the inhalations are administered through a glass tube held in the patient's mouth, the admixture of air and dilution of carbon dioxide take place; but the results by this so-called open method are quite satisfactory. The open method is recommended for patients who are markedly debilitated or who show some of the possible side-effects when the closed method is used. All patients taking 5 per cent carbon dioxide and 95 per cent oxygen use the closed method (B. L. B. mask).

It is a good policy to explain to the patient briefly the mode of action of the gas inhaled, and the expected changes in respiration, and the probable subjective symptoms. After proper instructions, inhalations through a glass tube can be administered without constant supervision; however, it is the responsibility of the nurse to regulate the flow of the gas, and time of the treatment. As a rule, the meter is set to 4 to 5 liters per minute for closed inhalations and to 5 to 7 liters per minute for the open method. The length of each treatment varies from 5 to 15 minutes; and the inhalations are administered once, twice, or three times a day. It is necessary to observe the patient closely during the first treatment. His respiratory response and subjective reactions determine the conduct of further treatments. They are conscious of breathing deeper and subsequent to the treatment they describe their experience in such terms as "the chest feels clear and cool"

and "the chest feels so much lighter". If it is noted that the respirations become too strenuous, the inhalations should be given with brief (1 minute) interruptions. In rare instances, when the closed method is used, it may be necessary to reduce the flow to less than 4 liters per minute. Most patients appear quite comfortable, as if in euphoria. The latter can be explained: (1) by the presence of 90 per cent oxygen in the gas mixture that is bound to counteract anoxemia; (2) by loosening up mucopurulent bronchial plugs and obstructive sticky, tenacious inflammatory exudate: the access of air is secured to underinflated regions of the lung; (3) by increased inspiratory expansion of the chest wall and by increased descent of the diaphragm atelectatic areas are stretched out and become aerated. In some of my patients I noted some transient minor side-effects of carbon dioxide inhalations, such as hot sensations, palpitation, weakness, frontal headache. and slight dizziness. None of these symptoms interfered with the treatment when proper adjustments were made in the method of administration. In the beginning, the treatments are given daily; subsequently, the frequency of inhalations can be reduced, depending upon the relief obtained. Some patients are obliged to take them daily for an extended period of time, while in others the interval between inhalations can be increased to a week. Carbon dioxide is an effective therapeutic agent, and in its use utmost individualization is required.

The prompt relief obtainable by this treatment is best expressed in the comments of patients: "the cough is not so dry, it is loose," "the cough does not jar me any more," "the cough is less, and not tight as before," "I do not have to exert myself when coughing," "I have no more dry spells of coughing," etc.

I have noted that following inhalations the amount of expectorated sputum is greater than before treatment, and also that adequate evacuation of the bronchi insures for the patient comparatively long periods of rest free of the annoying cough. Incidental by-effects of the satisfactory pulmonary drainage by carbon dioxide are: relief from dyspnea, undisturbed sleep during night, and improvement in the general subjective feeling. Often patients remarked how a feeling of pressure and heaviness was relieved by the treatment, that following inhalations their chest felt freer "like a loose sponge," and how much easier they were able to move about. Also I have noted the disappearance of chest noises and pharyngeal cough irritation.

When satisfactory evacuation of the bronchi has been accomplished, the amount of sputum becomes gradually less, unless further mucopurulent accumulation takes place during the interval

between treatments. It can be seen, therefore, that the frequency of inhalations and gas flow per minute have to be individualized and adapted to the changing requirements of the patient. I have found that the inhalation of carbon dioxide not only alleviates distressing cough but also enables one to reduce the consumption of narcotics and expectorant drugs.

The recent painstaking investigations of Basch, Holinger and Poncher[28] concerning the effectiveness of carbon dioxide and of the commonly used expectorants confirm our own clinical findings. They studied the influence of ammonium chloride, potassium iodide, fluid extract of senega, fluid extract of ipecac, and emetine hydrochloride, and compared it with the effect of carbon dioxide. They found that carbon dioxide acts as a real expectorant by diluting the sputum—that is, by lowering its viscosity and reducing its solid contents. They state that in comparing the physical and chemical properties of the sputum after the use of carbon dioxide inhalations with the same properties after the administration of drugs one at once realizes the greater liquefaction of the sputum caused by carbon dioxide: "since in this treatment there is no interference with the chemical properties of the sputum through the secretion of the administered drugs into it, the dried residue, the amount of ash and the total nitrogen content are regularly markedly lowered."

Striking results were observed by Alison[29] following the inhalation of carbon dioxide and oxygen in the treatment of acute bronchitis of infants and children. Satisfactory symptomatic relief was seen in bronchial asthma by Tiefensee,[30] Hurst[31] and Campbell and Poulton.[32] Gratifying symptomatic improvement was recorded in bronchopneumonia and in pneumonia in children by Grueneberg and Viethen,[18] and by Alison.[29] This procedure can be used with safety in bronchopulmonary infection of any etiology, including tuberculosis.

As to the selection of cases for this treatment, it is indicated whenever there is an accumulation and retention of inflammatory exudate in the bronchial tract and its evacuation—in spite of strenuous cough—is inadequate.

There are patients who should not be given this treatment: (1) patients with recent pulmonary hemorrhage; (2) those with marked emphysema; (3) when widespread pulmonary fibrosis is present without atelectasis, bronchiectasis or mucopurulent retention in the air-passages; (4) cases of acute plastic pleurisy and pleurisy with effusion; (5) hypertensive patients; and (6) when the cause of cough is outside of the lungs.

CONCLUSIONS

1. The rational management of cough depends upon its nature, origin, and upon the associated clinical findings.

2. Cough which originates from the lung may function with a high degree of failure (tussic insufficiency).

3. Ineffective cough harbors a number of potential dangers and, therefore, it should be corrected.

4. Suppression of the cough by depressing the cough reflex by narcotics, though easy to do, does not necessarily mean adequate treatment. As a matter of fact, there are a great many instances when the administration of narcotics may do more harm than good.

5. Unproductive cough may coexist with an accumulation of inflammatory exudate in the lower air passages.

6. No patient should be permitted to become exhausted under the strain of incessant coughing or to drown in his own accumulated pulmonary secretions.

7. Whenever considerable inflammatory exudate is present in the bronchopulmonary tract, relief from cough is best brought about by the adequate evacuation of these structures.

8. Drugs prescribed for the cleansing of the bronchi and alveoli should be selected according to the individual requirements of the case.

9. Expectoration is not the only means for the elimination of inflammatory exudates from the lung. Large amounts of the exudate are resorbed from the respiratory tract under favorable circumstances.

10. Clinical observation of others as well as my own experience have convinced me that carbon dioxide is a most efficient expectorant. It is superior to medicinal doses of potassium iodide, senega, ipecac and emetine hydrochloride. It is much better than steam inhalation. It aids the cleansing of the lungs both by facilitated expectoration and by pulmonary resorption.

11. When it is used as an expectorant, one can administer a mixture of 5 per cent carbon dioxide and 95 per cent oxygen, or 10 per cent carbon dioxide and 90 per cent oxygen. The strength of the gas mixture and the timing of its inhalation should be adjusted to the individual patient.

12. It is of advantage to combine carbon dioxide inhalation with steam inhalation.

13. Postural drainage may be instituted after the inhalation of carbon dioxide for the rapid evacuation of the respiratory tract.

14. Carbon dioxide by inhalation is given only as a symptomatic measure and it must not exclude specific drugs or accepted methods of treatment.

CONCLUSIONES

1. El tratamiento racional de la tos depende de su naturaleza, origen y hallazgos clínicos concomitantes.

2. La tos que se origina en el pulmón puede fracasar en su función (insuficiencia de la tos).

3. La tos ineficaz fomenta un cierto número de peligros potenciales y, por lo tanto, debe ser corregida.

4. La supresión de la tos mediante la depresión del reflejo de la tos por narcóticos, aunque fácil de lograr, no es necesariamente un tratamiento adecuado. En realidad, hay muchas ocasiones en las que la administración de narcóticos puede causar más mal que bien.

5. La tos improductiva puede coexistir con una acumulación de exudado inflamatorio en las vías respiratorias inferiores.

6. No se debe permitir que ningún enfermo se agote debido al esfuerzo excesivo de una tos incesante o que se ahogue en la acumulación de sus propias secreciones pulmonares.

7. Cuando quiera que exista una cantidad considerable de exudado inflamatorio en las vías broncopulmonares, la mejor manera de aliviar la tos es mediante la evacuación adecuada de ese material.

8. Las drogas que se receten para limpiar los bronquios y los alvéolos deben ser escogidas de acuerdo con los requisitos individuales del caso.

9. La expectoración no es el único medio de eliminar los exudados inflamatorios del pulmón. Cuando las circunstancias son favorables se reabsorben grandes cantidades del exudado en las vías respiratorias.

10. La observación clínica de otros, así como mi propia experiencia, me han convencido de que el anhídrido carbónico es un expectorante de lo más eficaz. Es superior a dosis medicinales de yoduro de potasio, sénega, ipecacuana e hidrocloruro de emetina. Es mucho mejor que la inhalación de vapor, y ayuda a limpiar los pulmones, tanto porque facilita la expectoración como por la reabsorción pulmonar.

11. Cuando se usa como expectorante, se puede administrar una mezcla de 5 por ciento de anhídrido carbónico y 95 por ciento de oxígeno, o 10 por ciento de anhídrido carbónico y 90 por ciento de oxígeno. La concentración del gas en la mezcla y la regulación del tiempo de inhalación deben ser adaptados a cada paciente.

12. Es ventajoso combinar la inhalación del anhídrido carbónico con inhalación de vapor.

13. Puede instituirse el drenaje de postura después de la inhala-

ción del anhídrido carbónico para facilitar la evacuación rápida de las vías respiratorias.

14. Se emplea la inhalación del anhídrido carbónico solamente como medida sintomática y no a exclusión de drogas específicas o de tratamientos aceptados.

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Surveys, B.C.G., Social Insurance and Health Card as Part of the Tuberculosis Control in Asuncion, Paraguay

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In July 1941, we started out in the organization of the Prophylaxis Section of our Tuberculosis Dispensary. By means of a thorough newspaper campaign, we were able to start our task by examining the near relatives of consumptive persons, of others who came to us by their own free will in a somewhat irregular manner, and the staffs of some public firms and private concerns, whose heads realized the importance of the work we proposed to carry out. In November 1941, the Ministry of Public Health realized the possibility of carrying out a collective examination. It amplified a law as of 1938 enforcing the use of the Health Card .... to make x-ray examination of the lungs obligatory for all public officials, public and private employees, teachers, students, professionals, as well as to those people who are about to travel.

Up to the time of writing this report we have taken x-ray photographs of 120,000 persons (1941-1946). The examinations consist of a tuberculin reaction, an x-ray photograph of the lungs and a brief exploration of the skin and mucous membranes. Occasionally and always with an investigational point of view in mind, biotypological data are taken such as complexion, height, weight, chest circumference, etc. Last year (1945) we have also given attention to the physical exploration of the abdomen. B.C.G. vaccination is given to all those persons who are tuberculin negative.

We can examine with ease 200 persons in three hours and the results of these examinations are delivered 48 hours later. In this lapse of time, it is possible to examine the x-ray photographs which were taken and to fill in the Health Card to be handed to the examined persons. When damage is found to exist in the heart and main arteries, or if it is necessary to carry out a further examination of any person within a certain time, the facts are entered on the respective cards. Any suspicious lesion of the lungs is checked and photographed by x-ray to make an accurate diagnosis. In such cases, the laboratories are instructed to take a blood

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count, blood sedimentation rate, and to examine the sputum. Studies of the gastric contents are rarely carried out. Instead we have introduced the bronchial washings (methods of Abreu and R. Fernandez from Brazil). In this technique, the posterior pharynx is anaesthetized with 0.1 per cent pantocaine usually introduced by atomizer spray. The tongue is retracted forward and about 10 cc. of saline is quickly injected into the posterior pharynx above the epiglottis. The ejected material is collected and examined for tubercle bacilli. We consider this procedure more practicable since it can be done at any time.

Those persons suffering from heart disease are urged to place themselves in the hands of private practitioners or of the public dispensaries. People suffering from lung disease are notified that they should report to the Tuberculosis Dispensary or at a hospital for treatment. When the patient is a worker or a private company employee, and he is found to be ill of tuberculosis, the Social Insurance Division is notified for the corresponding follow-up. The persons with syphilis are sent to the respective institutions for treatment. (In the finding of syphilis, the Chediack microreaction is used.) Leprosy cases are compelled to be interned in the Leprocomium at Sapucay, some fifty miles from Asuncion.

The results of our research have been printed in different publications.

SUMMARY

A Health Institute for Individuals and Communities has been established in Asuncion to provide health cards to all persons who carry out their part in the communal activities by means of their trade or profession. In order to obtain the health card, each person pays Gs. 2 ($.60) and undergoes an x-ray examination, a chest examination, a tuberculin test and examination of the skin and mucous membranes. B.C.G. vaccination is given to the tuberculin non-reactors.

Through these examinations we screen out the pulmonary, cardiac, venereal and leprosy cases. The card is not issued to people with other contagious diseases; such as, tuberculosis, syphilis in its contagious stage, leprosy, etc. In this way many people have learned for the first time that they were victims of such diseases and have promptly been treated for their respective ailments. This quick intervention and the consequent control that we exercise over the patients has allowed them to avoid what might eventually have led to irreparable damage to their health.

The Abreu method which is used is perhaps rather more expensive than the fluoroscope method but has proven to be highly superior in the number of pulmonary cases it has brought to light.

The importance of the Health Card is, therefore, herewith explained.

RESUMEN

Se ha fundado en Asunción un Instituto de Salud para Individuos y Colectividades, cuya función es proveer de tarjetas de salud a todas las personas que participan en las actividades de la colectividad por motivo de su negocio o profesión. Para obtener la tarjeta de salud cada persona paga Gs. 2 ($.60) y es sometida a un examen radiográfico, un examen del tórax, una prueba tuberculínica y un examen de la piel y de las membranas mucosas. Se vacuna con B.C.G. a los que no reaccionan a la tuberculina.

Mediante estos exámenes se descubren los casos pulmonares, cardíacos, venéreos y leprosos. No se les expide la tarjeta a personas que padecen de enfermedades contagiosas, tales como tuberculosis, sífilis en su período contagioso, lepra, etc. De esta manera muchas personas se han enterado por primera vez de que eran víctimas de esas enfermedades y han recibido pronto tratamiento para sus respectivas dolencias. Esta intervención oportuna y el control consiguiente que ejercemos sobre los enfermos les ha permitido evitar lo que con el tiempo habría podido causarles daño irreparable a la salud.

El método de Abreu que se emplea es, quizás, algo más costoso que el método roentgenoscópico, pero ha demostrado ser muy superior en el número de casos pulmonares que ha puesto al descubierto.

Se ha explicado, pues, la importancia de la Tarjeta de Salud.

Diasone Therapy in Pulmonary Tuberculosis*

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The sulfonamides have proved to be ineffective and disappointing in the chemotherapy of tuberculosis. Related chemical compounds, called sulfones, have recently been studied intensively and have been found to have therapeutic effect in experimental guinea-pig tuberculosis. The parent compound is 4,4′-diaminodiphenylsulfone. From this has been derived promin (sodium p,p′-diaminodiphenylsulfone-N,N′-didextrose sulfonate), promizole (4, 2′-diaminodiphenyl-5′-thiazolesulfone), and diasone* (disodium formaldehyde sulfoxylate diaminodiphenylsulfone).

It is our experience with the last mentioned drug which we now propose to report.

Method and Procedure:

Seventeen patients were selected for diasone therapy. Thirteen were white and four were negro. Eight patients were male and nine were female. Their ages varied from 19 to 36 years. Three had moderately advanced and fourteen far advanced pulmonary tuberculosis.

All had been at Koch Hospital for months, many for years. For the most part, conventional therapy, when indicated, had already been given. Thus, six patients had had phrenic nerve crushing, twelve one or more pneumothoraces attempted or established, and seven thoracoplasty.

Three of these patients who had had thoracoplasty were given diasone primarily because of persistently positive sputum. One patients' disease was actively spreading, and the remaining were patients who were not doing well, whose course was stationary or slowly becoming worse. All patients had a positive sputum. Most of them were on bed rest.

Our knowledge of these patients' course, our past experience with them and with patients with similar disease, we felt, were adequate controls.

Diasone was given by mouth, .33 grams three times a day with

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*Diasone was obtained for this study through the courtesy of Dr. George R. Hazel, of the Abbott Laboratories of Chicago, Illinois.

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meals, except to one patient who received it two times daily. Only rarely was it necessary to decrease or interrupt dosage because of toxic reactions. The length of treatment varied from 64 days to 387 days, the average being 202 days.

An x-ray film, physical examination of the chest, complete blood count, sedimentation rate determination (Wintrobe-Landsberg), and urinalysis were done prior to diasone therapy on each patient. During administration of the drug, a complete blood count, sedimentation rate determination, urinalysis, and diasone blood level determination were done weekly on each patient. X-ray films were taken at frequent intervals.

Results: A review of all cases after a course of diasone therapy showed:

No change	5 cases
Worse	6 cases
Died	1 case
Improved	5 cases

Three of the cases listed as worse later died, 102 days, 51 days, and 18 days subsequent to the discontinuation of the diasone. These patients had received diasone for 125 days, 106 days, and 64 days, respectively.

The following is an analysis of the cases which, in some way or another, seem to have shown some improvement:

1. Patient V.V., a 38 year old white male, whose pulmonary tuberculosis was diagnosed Far Advanced II on admission. There was bilateral cavitation. A left pneumothorax and intrapleural pneumonolysis were followed by a right thoracoplasty. Subsequently the left pneumothorax was lost, but the patient improved, and was started on exercise. While on exercise, the left lung showed increased disease. The patient was then given diasone. Five months after diasone was begun the patient had a hemoptysis. An emergency pneumoperitoneum was instituted. The patient has done fairly well since then. There is some improvement on x-ray. The sedimentation rate has dropped from 24 mm. (corrected) to 13 mm. (corrected). The Schilling differential count has remained about the same. There has been no fever. The sputum remains positive. The improvement here is only slight, and might be due to the pneumoperitoneum.

2. Patient H.C., a 33 year old male, whose pulmonary tuberculosis continued to spread despite a left pneumothorax, which was ineffective, and a left phrenic nerve crushing. After the first stage of thoracoplasty, his disease spred to the right lung, necessitating delay in thoracoplasty. He was then given diasone. Subsequently he improved, and thoracoplasty was completed while he was receiving diasone therapy. Since then his improvement has been definite, but this may be due, at least in part, to the thoracoplasty.

3. Patient D.S., a 25 year old negro male, who had had a right thora-

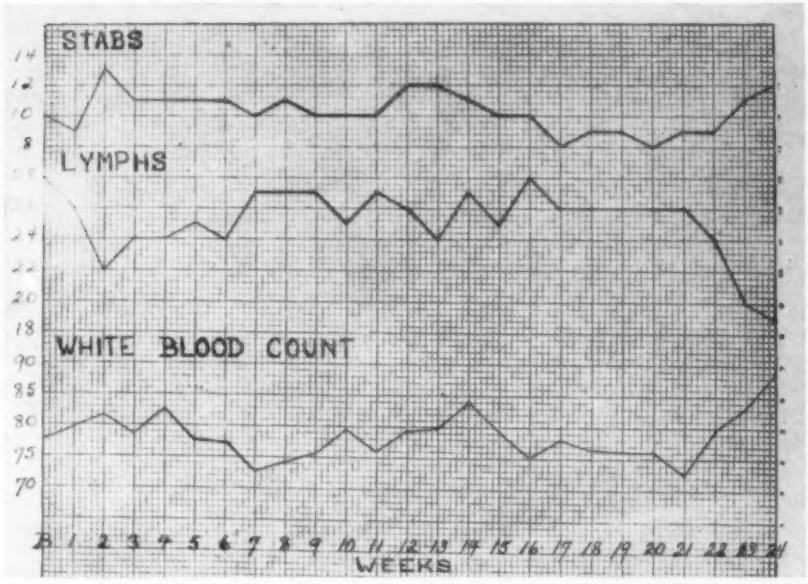

Figure 1: A composite graph of the "stabkernige" cells, lymphocytes and the white blood counts of all patients taken weekly. In the case of the stabs and lymphocytes the figures in the ordinate represent percentages and in the white count hundreds per c.mm.

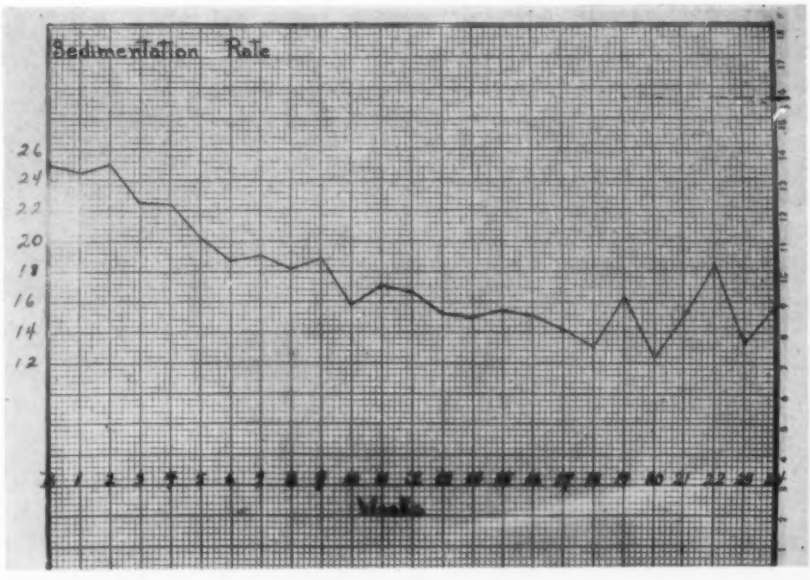

Figure 2: A composite graph of sedimentation rates in mm. per hour (Wintrobe-Landsberg) of all patients taken at weekly intervals.

coplasty, but whose sputum remained persistently positive. Bronchoscopy in July, 1944 revealed the presence of tuberculous ulcerations of the left main bronchus, which were cauterized with 30 per cent silver nitrate. Diasone therapy was then begun. In the following October, one sputum specimen was positive out of 10, and in December, 1944, ten sputum examinations were negative. Bronchoscopic examination at this time, however, showed that extensive ulceration of the left main bronchus was still present. Diasone was then discontinued. The ulcerations were then repeatedly cauterized with 30 per cent silver nitrate. Later bronchoscopic examinations showed some improvement. Sputum examinations have continued to be negative.*

4. Patient A.W., a 33 year old white female, who had a left phrenic nerve crush followed by a right pneumothorax. The latter had to be discontinued because of dyspnea. At the time that diasone was first given, there were heavy infiltrations bilaterally, a large cavity on the left below the clavicle, and a shallow cavity on the left at the level of the fourth interspace anteriorly. After a course of treatment with diasone, the cavity in the left apex became smaller, but the cavity in the left mid-lung field became larger. In general, however, the entire picture appeared somewhat improved.

5. Patient M.L., a 40 year old white female, diagnosed on admission as pulmonary tuberculosis, Far Advanced, II, with bilateral cavitation. Cholecystograms demonstrated the presence of gallstones. Following administration of diasone, she felt subjectively stronger and gained 16 pounds. There has been only a small amount of bilateral improvement on x-ray examination, however, and there is one new area of involvement in the left apex which appeared during treatment. There has been a change in the Schilling differential count, from 2 stab forms and 21

*Since this paper has been written, this patient has again developed positive sputum, approximately 8 months after discontinuing diasone.

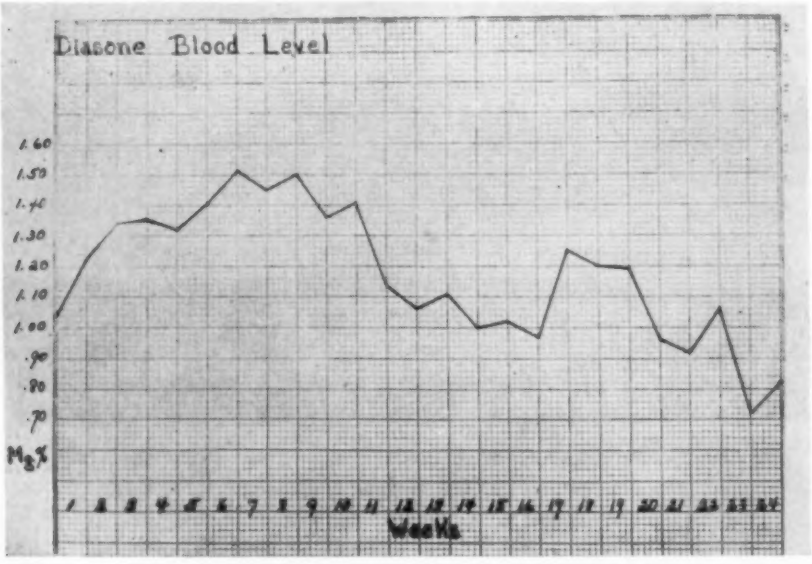

Figure 3: A composite graph of diasone blood levels taken on all patients at weekly intervals.

lymphocytes before diasone to 8 stab forms and 26 lymphocytes after diasone. The sedimentation rate has dropped from 46 mm. to 32 mm. (corrected). It seems to us that the curve of her improvement has recently levelled off.

Figure 1 is a composite chart, showing the variation in stab forms, lymphocytes, and the white cell count on all patients under therapy. There seems to be no definite trend in any one direction.

Figure 2 is a composite chart showing the improvement in sedimentation rate. This is definite, and often, it appears to us, is out of proportion to the actual improvement in the patient.

Figure 3 is a composite chart showing the variation in the diasone blood level. For the most part, the levels were between 1.0 and 1.5 mg. per cent. These are about the same levels obtained by Petter and Prenzlau[5] and Benson and Goodman.[6]

Toxicity:

The most consistent toxic effect of diasone was the development of anemia (Fig. 4). The average red cell count before diasone therapy was 4,348,000 per c.mm. At the fifth week after diasone therapy the average red cell count was 3,760,000. This is an average loss of 588,000 red cells per c.mm. Following this, there was a slow recovery which almost, but not quite, reached the level before drug therapy was begun.

The curve of hemoglobin (Sahli) followed that of the red blood count.

The next most common toxic effect was cyanosis, which was

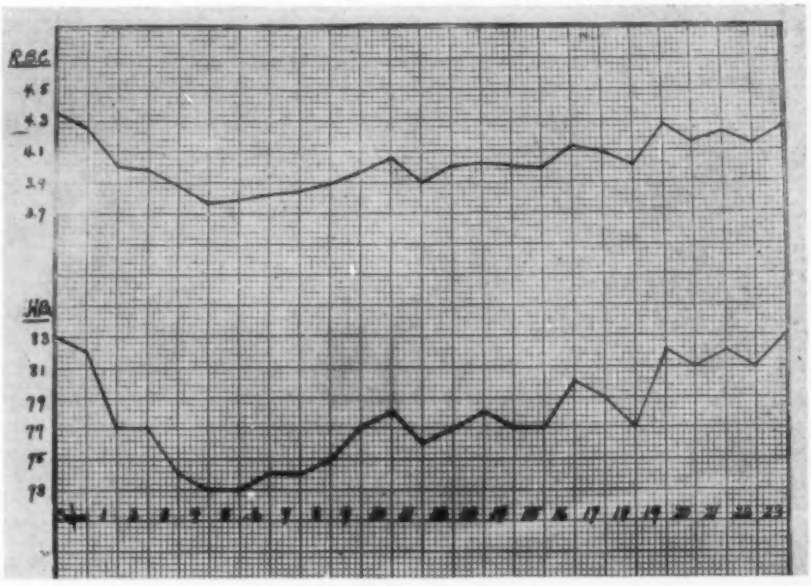

Figure 4: A composite graph of red blood counts in millions per cu. mm. and hemoglobin percentages of all patients taken at weekly intervals.

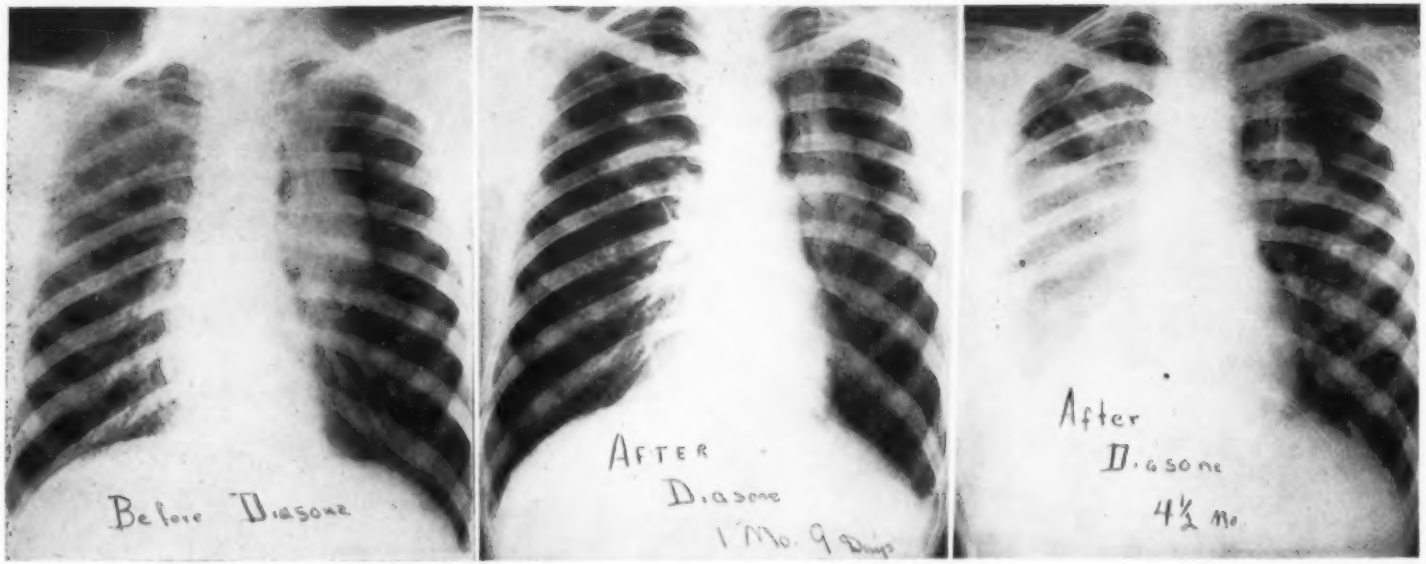

FIGURE 5 a FIGURE 5 b FIGURE 5 c

Fig. 5 a: (Patient W.P.) X-ray of chest just before diasone therapy, showing left pneumothorax.—*Fig. 5 b:* (Patient W.P.) X-ray of chest 1 month, 9 days after beginning of diasone therapy, showing bilateral pneumothorax.—*Fig. 5 c:* (Patient W.P.) X-ray of chest 4½ months after beginning of diasone therapy, showing extensive bilateral hematogenous spread.

seen in 12 of the 17 patients. This persisted throughout the administration of the drug, but disappeared after diasone therapy was discontinued.

Other toxic effects noted were:

Nervousness	5 cases
Nausea	4 cases
Malaise	4 cases
Headache	3 cases
Tremor	2 cases
Vomiting	2 cases
Weakness	1 case
Diplopia	1 case
Blurred Vision	1 case
Dyspnea	1 case

These toxic effects seemed unrelated to the diasone blood levels.

We had no cases of dermatitis. Exfoliative dermatitis[6,8] and a fatal pemphigoid reaction to diasone[10] have been reported.

There had been no evidence of impaired hepatic or renal function. In one patient, a vaginal discharge (non-specific), cleared during diasone therapy.

One patient, W.P., died while under diasone therapy. He was a 22 year old white male whose pulmonary tuberculosis was diagnosed Far Advanced III on admission. Because of severe pharyngeal and laryngeal involvement his prognosis was considered very bad, and active therapy was instituted. A left pneumothorax was begun (Fig. 5-a), followed 2 months later by a right pneumothorax (Fig. 5-b). Just before the institution of the right pneumothorax, a course of diasone was begun. Three months later it was felt that his condition was stationary, except for rather definite improvement in his pharyngeal involvement. One month later the patient had a miliary spread bilaterally (Fig. 5-c). The patient died while receiving diasone therapy, after 5 months of treatment.

Post-mortem examination as made by Dr. John Saxton and Dr. Adrian Neerken was reported as follows:

> The body is that of a 22 year old white man weighing an estimated 115 lbs. and being an estimated 69 inches in length. The body is markedly undernourished but development appears normal.
>
> *Peritoneal Cavity:* Upon opening the peritoneal cavity the general disposition of the viscera is normal. The serosal surfaces are smooth and glistening.
>
> *Pleural Cavity:* There is a moderate degree of collapse of both lungs with an increased amount of free pleural space. The visceral pleura is thickened bilaterally.

Pericardial Cavity: The pericardium is thin and the cavity contains an estimated 10 cc. of straw-colored fluid.

Thyroid: Parathyroids: Not remarkable.

Thymus: Not identified.

Larynx: The larynx is opened and shows an ulcerating lesion on both vocal folds and on the epiglottis. The borders of these ulcerations are slightly raised, are dirty and ragged.

Heart: The heart weighs an estimated 330 grams. It is of the usual shape and color. The endocardium is smooth and glistening. The mycardium is red-brown in color and appears in good condition. The foramen ovale is anatomically closed. The heart valves are thin and appear competent. The coronary vessels are patent throughout.

Vessels: The aorta shows a smooth intimal surface. The vessels show a marked elasticity. The remainder of the blood vessels are not unusual.

Lungs: The pleural surfaces have been described. Palpation reveals a nodularity throughout both lungs. The cut section shows several cavities in both apices, the largest being approximately 3 cm. in diameter, and containing yellow-green foul pus. Throughout both lungs many small caseous lesions varying from 1 mm. to 5 mm. in diameter are seen. Many of these are clover leaf shape. They are firm and slightly raised and the centers are caseous. There is a marked dissemination throughout both lung fields. The tracheo-bronchial lymph nodes show many small caseous areas. The trachea and bronchi themselves show an injection of the submucosal vessels but otherwise are not grossly remarkable.

Spleen: The spleen weighs an estimated 200 grams. The capsular surface is slate-gray in color. Upon cut section many gray follicles are present. The splenic parenchyma is soft and purple red in color.

Alimentary Tract: The alimentary tract is examined from mid-esophagus to anus. No gross lesion is identified.

Pancreas: The pancreas is of the usual size, shape, and consistency. Upon cut section the typical gray lobules are noted.

Liver: The liver weighs an estimated 1200 grams. The capsular surface is smooth. Upon cut section the usual vascular markings stand out with slightly increased intensity, giving a nutmeg appearance to the cut surface.

Biliary Tract: The gall bladder is of the usual pear shape and contains an estimated 15 cc. of viscid, red-brown bile. The biliary tree is patent throughout.

Adrenals: Not remarkable.

Kidneys: The kidneys weigh an estimated 150 grams each. The capsules strip without difficulty revealing a smooth cortical surface. On cut section the usual cortico-medullary ratio is present. An occasional small white caseous area is present in the region of the medulla. The mucosa of the renal pelvis is smooth.

Ureters and Urinary Bladder: Not remarkable.

Reproductive Organs: The external genitalia have been described. The testicles are not unusual. Upon sectioning the prostate a small caseous area approximately 1 cm. in diameter is present in one lateral lobe. Aside from this the prostate is not remarkable.

Lymphatic System: Not remarkable.

MICROSCOPIC FINDINGS

Heart: No significant change is present in the section studied.

Larynx: A section through the larynx shows an infiltration of the

submucosal portions with many mononuclear cells and lymphocytes. In several areas the epithelium is absent and there is evidence of caseation. A large cartilage is being eroded.

Lung: The study of sections from various portions of the lungs shows a widespread dissemination of poorly formed caseous tubercles. There is little epithelioid proliferation noted, and Langhans giant cells are rare. Other apparently healed, fibrotic nodules are also seen. In several areas the alveoli contain edema fluid and polymorphs as seen in bronchopneumonia. No evidence of bronchial tuberculosis is seen.

Liver: Throughout the liver a number of small fibrous nodules are present. These are surrounded by a lymphocytic infiltration in many areas, and are more proliferative than caseous in character.

Kidneys: Sections of the kidneys show an occasional fibrous tubercle in the cortex. A rather large caseous area is present in the medulla of one kidney, surrounded by epithelioid cells and lymphocytes. Only an occasional Langhans giant cell is seen.

Prostate: In the lateral lobe several large tubercles are noted. There were no changes in the histological sections of liver, spleen or kidneys which could be ascribed to diasone.

Final Diagnosis: Lungs: Fibrocaseous Tuberculosis, Advanced with Cavitation, Bilateral; Tuberculosis of Larynx, Kidneys, Prostate, Liver; Bronchopneumonia.

Discussion:

In 1941 Hinshaw and Feldman[1] and Hinshaw, Feldman, and Moses,[2] reported very favorable results in the treatment of experimental tuberculosis in guinea-pigs with promin.

Later Callomon[3] confirmed this work, but found that diasone produced almost as favorable results, and was less toxic. Later Feldman, Hinshaw, and Moses[4] also studied the effect of diasone in experimental tuberculosis and corroborated these results. All of these studies were carefully done, and not only were there marked differences in mortality rate, but striking differences on pathological examination between the treated and untreated animals.

In 1944, Petter and Prenzlau[5] treated 44 patients having tuberculosis with diasone and found that 100 per cent of minimal and moderately advanced cases, and 78 per cent of far advanced cases showed some degree of improvement. Fifty-nine per cent of their cases showed sputum conversion and 43 per cent the closure of cavities.

Less encouraging results were reported by Benson and Goodman[6] who found only four cases out of 22 patients so treated whom they felt may possibly have benefited from the drug.

Recently Pfuetze and Pyle[7] reported treating 36 patients with diasone. They felt that 19 cases showed some improvement, and 17 were either unchanged or worse.

Five of our seventeen cases showed some improvement. None of these changes was remarkable or spectacular. In three of the

cases, other procedures may have played a part. In one case, although improvement has occurred in one area, in another area a cavity has increased in size. In the last case after initial improvement, the clinical course has remained stationary.

Corper and Cohn[9] feel that the mode of action of diasone in experimental tuberculosis may be largely through anoxemia. They also feel that since 1 gram of diasone per day has not caused evident anoxemia in man, that it is probable that therapeutic dosages have never been obtained. It is also highly probable, they say, that such therapeutic dosages would be dangerous.

The test which we imposed on diasone as a chemotherapeutic agent was a difficult one. The seventeen cases were mainly those in which ordinary therapy had failed. We did not try to determine whether diasone had any effect whatsoever on the course of human tuberculosis, but rather whether it has an effect significant enough to consider it a worthwhile clinical therapeutic adjunct.

Hinshaw and Feldman[1] caution against the "drawing of either positive or negative conclusions . . . until the force of facts makes these conclusions self evident." To this we agree.

Dr. J. Arthur Myers, in discussing their paper speculated upon the time "when a person who is found to react to tuberculin, but who has no evidence of tuberculosis will be treated by chemotherapy, just as the patient who has a positive Wasserman with no other evidence of syphilis is now treated by chemotherapy."

This time may come to pass. But we do not believe that the drug will be diasone.

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Bronchiectasis: A Neglected Disease

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Within the past quarter century our knowledge of bronchiectasis has been enormously advanced. Iodized oil bronchography, introduced by Sicard and Forestier[12] in 1922, has been developed into a precise and easily applied method of diagnosis. Its wider use has shown that bronchiectasis is a very common disease, second only to tuberculosis as a chronic disease of the lungs in the opinion of many, even more common than tuberculosis in the estimation of others. Surgical treatment has been developed to the point that cure in properly selected cases is practically certain and operative mortality is strikingly low. Thus, a disease once considered uncommon, incureable and susceptible only to palliative medical treatment, is now known to be very common, is cureable, and in some of its manifestations belongs just as surely in the realm of surgical disease as does acute appendicitis.

These events comprise one of the major advances in the field of medicine, an advance little noted except by physicans who are particularly interested in diseases of the chest, and even these have been hard put to it to keep abreast of the times, so rapid have been developments in the field of chest surgery.

During my years in private practice I saw little evidence that other members of the profession in the community where I practiced were aware of the commonness of bronchiectasis, and I myself would not have been impressed by its frequency had I not served on the staffs of two tuberculosis sanatoria where I saw many cases admitted with the mistaken diagnosis of tuberculosis.

As a naval medical officer it was most emphatically impressed on me that many cases are being taken into the armed forces because of the lack of awareness on the part of recruiting physicians of the symptoms of bronchiectasis or of its frequency. Other cases are developing after entrance into the service but go undiagnosed because of a lack of knowledge of how bronchiectasis develops or of the signs and symptoms that should make one think of it. Among the patients whom I have diagnosed as having the disease I have rarely found one who has so much as heard the word *bronchiectasis*. And when it is proposed that a part of a lung shall be removed he is completely overwhelmed by his fear of the consequences.

It is evident that a program of education concerning bronchiectasis is in order.

MATERIAL

During the eighteen months that I was on the general chest service in a naval hospital, a diagnosis of bronchiectasis was made by means of iodized oil bronchograms in 59 cases. These patients comprised .6 per cent of the 9754 cases admitted to the hospital during that period, were the largest group of all the chronic non-tuberculous chest diseases treated on the service. Fifty-three of these patients were ultimately discharged from the Navy and comprised 4.8 per cent of the 1102 cases discharged during the same period.

Of these 59 patients only six had had a diagnosis of bronchiectasis previous to their admission to our service. In these six cases the disease was of many years standing and the symptoms "typical". In two other cases the diagnosis had been suggested but rejected because the patients were considered not to have the characteristic symptoms of bronchiectasis. As one medical officer put it, "This patient does not have the typical expectoration of large quantities of foul sputum."

Of particular interest to me was a group of 40 patients admitted to my ward at one time. They were part of a group of patients sent from a large naval training camp in the Northwest for further convalescence from complications or sequelae of streptococcus pneumonia, contracted during a severe epidemic of streptococcus infections that swept the camp. They were selected at random for admission to my ward and therefore were a fair sample of the entire group.

Of the 40 patients, 19 had only thickened pleura following empyema, one had chronic empyema, and 20 had residual pulmonary infiltrations which had persisted for approximately three months from the time of onset of the acute illness. Iodized oil bronchograms done on these latter 20 cases showed bronchiectasis in 12 of them. In the remaining eight cases the infiltrations ultimately disappeared.

Thus, of a group of cases with sequelae of streptococcus pneumonia, 30 per cent of the entire group were shown to have bronchiectasis and of the subgroup with persistent infiltrations in the lungs, so-called "unresolved pneumonia," 60 per cent were shown to have bronchiectasis.

ANTECEDENT ILLNESSES

In the histories of the entire group of my patients, pneumonia was given as the antecedent illness in 29 cases, upper respiratory infection (rhinitis, sinusitis, pharyngitis, tonsillitis) in 15, asthma in 2, scarlet fever in 2, whooping cough in 2, measles in 1, lung abscess in 1 and inhalation of poison gas in 1. In six cases the

antecedent illness was unknown, the patients usually stating, "I have had a cough ever since I can remember."

It is probable that in most cases of bronchiectasis an area of pneumonia will be found at the inception of the disease if a chest roentgenogram is made. In going over the records of this series of cases it was found that in most instances, when the patient was seen in the dispensary before a chest roentgenogram was made, the diagnosis was of some type of upper respiratory infection, or acute catarrhal fever, but after admission to the hospital and the making of a roentgenogram the diagnosis was usually changed to bronchopneumonia, or atypical pneumonia.

There were a number of cases in the group following acute upper respiratory illnesses in which the symptoms of the antecedent illness were no more severe than those of a bad head cold. The history that so often is heard was given of a cold that "settled on the lungs." The only thing to suggest bronchiectasis was the persistence of cough and expectoration.

In only seven cases of this series was there evidence of purulent sinusitis during our observations of the patients. In five of the cases the purulent discharge was transient and in only two was it persistent enough to require prolonged treatment.

In recent years there has been a general trend away from regarding sinusitis as of etiological importance in the development of bronchiectasis. Most authorities now regard it as only coincidental. With this view I heartily agree.

AGE AT ONSET OF SYMPTOMS

The age at the onset of symptoms given by the patients is tabulated by decades in table 1. These figures agree with those of other observers which show that bronchiectasis has its onset in most instances during the first two decades of life. When it has its onset in later life some condition causing bronchial obstruction such as tumor, foreign body and asthma must be thought of in addition to the aforementioned causes.

TABLE 1: AGE OF ONSET BY DECADES

Decade	*Number*	*Per cent*
1 - 10 years	16	27.1
11 - 20 years	18	30.5
21 - 30 years	19	32.2
30 - 40 years	6	10.2
TOTAL	59	100.0

DURATION OF SYMPTOMS

The length of time that the patients in this study had had symptoms is shown in table 2. It is of interest to note that 24 of our patients gave as the duration of their symptoms six months or less and an additional five, a year or less. Of the group of 24 cases, all had had streptococcus pneumonia in the large naval training camp previously mentioned. In all of these cases symptoms had been present approximately three months at the time we first saw them. However, it must be emphasized that their symptoms had been continuous ever since their attack of pneumonia. This is an important point. Most physicians think of its chronicity as being one of the most characteristic aspects of bronchiectasis. But most often bronchiectasis has its inception during an acute illness, usually bronchopneumonia, and it is therefore reasonable to conceive of it as having its acute, subacute, and chronic stages. Some authors have gone so far as to speak of "pre-bronchiectasis". To forget all but the chronic stage is to help bring about the situation described by Hinshaw and Schmidt,[7] who say, "Most of our patients who have bronchiectasis have had unquestionable symptoms for many years before a diagnosis was made. Often the disease has progressed to such an extent that it is incureable."

SYMPTOMS

Cough and expectoration occurred in every case. In many instances the cough was not severe and the expectoration did not exceed one-half to an ounce of sputum raised in the period im-

TABLE 2: DURATION OF SYMPTOMS

Time interval	*Number*	*Per cent*
0 - 6 months	24	40.7
7 - 12 months	5	8.4
1 - 2 years	3	5.1
2 - 3 years	1	1.7
3 - 4 years	2	3.4
4 - 5 years	1	1.7
5 - 10 years	9	15.4
10 - 15 years	5	8.4
15 - 20 years	4	6.8
20 - 25 years	5	8.4
TOTAL	59	100.0

mediately after arising. In some of the cases there was complete absence of cough or expectoration for variable periods. *Foul sputum* occurred in but two cases and in these cases was only mildly foul. Joress and Robins[11] found but two cases with foul sputum in a group of 32 cases of bronchiectasis diagnosed at an army hospital. It is evident from these observations and from the findings of other clinicians that foul sputum is not of sufficient frequency to be considered of important diagnostic significance in bronchiectasis.

Increased susceptibility to respiratory infections was complained of by the majority of the patients. Bouts of acute illness characterized by malaise, fever of one to a few days duration, and increased cough and expectoration, were common. In most instances such illnesses, prior to the patient's enlistment in the navy, had been diagnosed influenza, or occasionally, pneumonia.

Hemoptysis and pulmonary hemorrhage were presenting complaints, or occurred during our observation of these patients, in 19 cases or 32 per cent. Bronchiectasis is one of the most frequent, but at the same time the least frequently thought of, cause of pulmonary bleeding.

Dyspnea with exertion was complained of by six patients. These included the two cases who had suffered from asthma prior to their developing symptoms of bronchiectasis, one other case that developed asthma subsequent to the onset of his symptoms of bronchiectasis, and three cases with disease of many years standing. In all cases of bronchiectasis of long standing there is significant pulmonary fibrosis and emphysema. In certain instances, these changes, in addition to pulmonary arteriosclerosis which accompanies them, ultimately lead to right heart failure.

Lack of energy, weakness, and lassitude were complaints in a few cases but there was no detectable correlation between these symptoms and the extent of the bronchiectasis in any given cases.

PHYSICAL FINDINGS

Medium moist rales in the lungs, due to the pneumonitis that commonly accompanies bronchiectasis, were heard in all of these cases except three. In many cases *coarse bronchial rhonchi* were also heard. In those cases with areas of atelectasis or pneumonitis significantly large, *dullness and changes in the breath sounds* over the involved area were detected.

Clubbing of the fingers was found in but one of our cases. In a series of 32 cases found by Joress and Robins[8] in an Army station hospital, this finding was present in but one case. This low incidence of clubbed fingers is in marked contrast to that reported by Findlay and Graham[4] who found it in 50 per cent of their cases,

by Ogilvie[10] who found it in 39 per cent, or Fletcher[5] who found it in 35 per cent.

Lisa and Rosenblatt[9] state, "The clubbing of digits is an important sign in bronchiectasis and occurs in a significant percentage of cases. . . . There is no direct relationship between the extent or duration of bronchiectasis and the incidence of clubbing. . . ."

With the latter statement I cannot agree. The writers who report a high incidence of clubbing of the fingers are probably reporting on cases of long standing. In my opinion when clubbed digits are present, there are always other signs that make the diagnosis fairly obvious.

RADIOGRAPHIC FINDINGS

The types of x-ray shadows found on the standard film made at the time of admission of these patients to our service, is summarized in table 3. *Thickening and fuzziness of the linear shadows* in the lower lung field, was in this series, as in most others, the most frequently encountered radiographic abnormality.

A common error in interpretation of this type of shadow is to consider the normal vascular shadows which are heaviest in the bases along the heart borders—precisely the same locations where abnormal peribronchial infiltration is usually found—as being abnormally thickened linear markings. If there is any movement at all during the making of the chest roentgenogram, these shadows are certain to be fuzzy, which increases the illusion that they are abnormal. Therefore, all films of the chest, in order to be of proper technical quality, must be made with a short enough exposure time that all movement is "frozen".

Atelectasis may be associated with bronchiectasis, and was evident in the chest roentgenogram in nearly half of our cases. It is often not apparent on superficial examination. The commonest site for bronchiectasis is in the posterior divisions of the lower lobe bronchus, behind the heart. If atelectasis occurs in such an area the increased density of the lung tissue is hidden by the heart or diaphragm, and manifests itself by a shift of the former to the left and an elevation of the latter. These changes in position are often not great and must be carefully looked for. Serial films, by showing a shift of the heart to the midline or of the diaphragm downward, may provide the only clue that atelectasis was previously present. In only one of our cases was seen the triangular basal shadow in the cardio-phrenic angle, characteristic of lower lobar atelectasis.

Pneumonic shadows were seen in the roentgenogram of approximately one-third of our cases. In all but two instances these were

of patchy, flocculent appearing type, and were usually interpreted by the roentgenologist as representing bronchopneumonia or, less commonly, atypical pneumonia.

Ring shadows, which Andrus[2] saw in a large percentage of his cases and which he ascribed to localized emphysema, were seen in 21 per cent of this series. In my opinion, the significance of this type of shadow is not easy to determine and I do not consider them of much importance in helping to make a diagnosis of bronchiectasis. They are invariably accompanied by shadows of more significance.

It must be emphasized that bronchiectasis *per se* causes no abnormal x-ray shadows. The latter are due to the accompanying pneumonitis, atelectasis, and fibrosis. In a significant number of cases, 18 per cent of this series, a *negative roentgenogram* was reported. Bronchograms are always necessary for positive diagnosis.

TECHNIQUE OF IODIZED OIL BRONCHOGRAPHY

Many techniques for the introduction of iodized oil into the bronchi have been described, but for general use there is but one practical method: the supraglottic technique. It is simple, requires no special apparatus and causes a minimum of discomfort to the patient.

The success of the method depends largely upon the use of adequate sedation and topical anesthesia prior to instillation of the oil. I use 15 mg. (¼gr.) of morphine and 0.45 mg. (1/150 gr.) of atropine hypodermically one hour before the examination is made. Immediately before instilling the oil the pharynx is thoroughly sprayed with a 2 per cent solution of pontocaine. The atomizer tip is then directed downward and, as the patient inhales, the spray is directed into the larynx and trachea. For most cases this amount of anesthesia is sufficient. For some patients, whose cough reflex is particularly sensitive, a cc. or two of pontocaine solution may be dripped into the larynx through the same cannula that is later used for the iodized oil.

The iodized oil, which has previously been warmed to body tem-

TABLE 3: TYPES OF X-RAY SHADOWS

Type of shadow	*Number*	*Per cent*
Thickening and fuzziness of the linear markings	46	78
Evidence of atelectasis	28	48
"Ring shadows"	12	21
Pneumonic shadows	19	30
No abnormal shadows	11	18

perature in a warm water bath, is introduced into the pharynx through a long cannula attached to a 10 cc. syringe. During this procedure the patient grasps the tip of his tongue with a piece of gauze and pulls it firmly forward. I feel that concentrating his attention upon this simple act, rather than on the manipulations of the operator in his throat, prevents the patient from gagging. It makes no difference into what part of the pharynx the oil is introduced, it will flow into the larynx and thence into the trachea unless the patient coughs, gags, or swallows.

The oil is directed into the bronchi which the operator desires to visualize, by proper positioning of the patient. For routine work the patient is placed in four positions: (1) with his thorax inclined forward and to the right, (2) backward and to the right, (3) forward and to the left, (4) backward and to the left. These positions permit visualization of the bronchi of both lower lobes, the right middle lobe and the lingula of the left upper lobe. It is only in exceptional cases that visualization of the right upper lobe and of the left upper lobe, with the exception of the lingula, is required. If this is necessary, I have found that it is easily accomplished by instilling iodized oil into the lower lobe of the homolateral lung and then placing the patient upon an inclined plane with the upper lobe which is desired to be visualized in the most dependent position. The most satisfactory inclined plane is of course a fluoroscopic tilt table. The flow of oil from the lower lobe into the upper lobe bronchi can then be watched and when it is satisfactory, roentgenograms made.

Routinely in bronchography, the roentgenograms that are made comprise posterio-anterior, and lateral views. It is also desireable to have oblique views. These "spread out" the shadows of the lower lobronchi and facilitate their study. For my part, if the number of films that can be made is limited, I prefer a stereoscopic pair of posterio-anterior films.

Interpretation of the results of bronchography. If satisfactory filling of the bronchi to be visualized is obtained, the presence or absence of bronchiectasis is usually obvious. Dilatation of the diseased bronchi is usually sufficient so that it cannot be overlooked. Since the diseased bronchi have lost their ability to eliminate foreign material, the opaque oil fills them more solidly than it does the normal bronchi, which begin to eliminate the oil immediately and soon contain but a coating of it on their walls. Furthermore, the oil readily penetrates the alveoli connected with normal bronchi but not those connected with ectatic bronchi. The result is what has been labelled a "leafless tree" appearance. The diseased bronchi appear as the broken stump of dead limbs on a dying tree.

However, there are cases in which the bronchial changes are so inconspicuous that it is difficult to determine whether they represent normal variations in calibre or beginning bronchiectasis. It must be remembered that the diminution of the calibre of bronchi, as one passes from the larger to the smaller divisions, is very uniform and if one finds any "beading" or localized bulging along the course of a bronchus it is entirely likely that it represents early bronchiectasis.

The location of the lesions in this series, as shown by bronchography, is summarized in table 4. In this, as in most reported series, the commonest location for bronchiectasis is the left lower lobe, and here, the bronchi in the posterior portion behind the heart are those most often involved. In this area advanced bronchiectasis may be present without there being any evidence of it on a standard roentgenogram of the chest.

It must be noted that in 41 cases (70 per cent) of this series the disease was unilateral and that in most of these (40 cases) it was confined to one lobe. This finding is in marked contrast to that reported by Churchill[3] that in 80 per cent of the cases operated on by him in which the left lower lobe was removed, the lingula of the left upper lobe was found to be involved also, and to the statement by Alexander[1] that, "The lesions are more often bilateral than unilateral." Probably the explanation of this discrepancy is that 49 per cent of our series of cases were diagnosed when symptoms had been present for less than a year. The involvement of the bronchi corresponded to the original area of pneumonia.

TABLE 4: LOCALIZATION OF LESIONS

Area of involvement	*Number*	*Per cent*
Left lower lobe alone	23	39
Right lower lobe alone	13	22
Both lower lobes	12	20.3
Right middle lobe alone	2	3.4
Both lower lobes and right middle lobe	2	3.4
Both lower lobes, right middle lobe and lingula of left upper lobe	2	3.4
Right upper lobe alone	1	1.7
Lingula left upper lobe alone	1	1.7
Right middle and right lower lobes	1	1.7
Left lower and right middle lobes	1	1.7
Both lower lobes and lingula of left upper lobe	1	1.7
TOTAL	59	100.0

Though there is a widespread impression that bronchiectasis is a progressive disease, there is no unanimity on this point. Lisa and Rosenblatt[9] feel that the maximum damage is usually done at the time of onset of the disease and there is little tendency for it to increase in severity or extent. Churchill[3] says, "We have to admit that bronchiectasis, under observation, progresses slowly if at all."

It must be admitted that there is no close correlation in any individual case between the length of time symptoms have been present and the extent and severity of the bronchiectasis. A patient may have had his disease for 25 years and still have unilobar bronchiectasis or may have had it for a short time and show multilobar involvement. Nevertheless, it was a very conspicuous fact that, in this series, those who had the least bronchiectasis practically all fell in the group of 24 cases who had had streptococcus pneumonia in the large training camp three months previous to admission to our service. To me it is inconceivable that an active infection in the lung, rendered incureable by irreversible tissue changes, should remain over a period of years, confined strictly to the area of original involvement. The frequency of flare-ups of acute pneumonitis testifies that the adjacent lung tissue is not capable of developing any effective immunity.

For my part, I shall consider as a safe working principle that the earlier bronchiectasis is diagnosed, the less the involvement that will be found, and therefore the greater the certainty that the patient can be cured.

COMPLICATIONS OF BRONCHIECTASIS

The occurrence of episodes of *acute pneumonitis* is so constant in patients with bronchiectasis that it can logically be considered a part of the disease syndrome. Sometimes the parenchymal involvement becomes so extensive as to result in lobar consolidation. Such an episode is not infrequently the final one in a patients' illness. *Pulmonary abscesses,* usually of small size are probably present in all cases of bronchiectasis. Those of such size as to be diagnosable roentgenographically are not so frequent. Extension of the pulmonary suppuration to the pleural space resulting in *empyema* is not uncommon. If the disease is of long enough standing *pulmonary fibrosis and emphysema* are inevitable. These changes may become so severe as to lead to *cor pulmonale. Amyloidosis* is always listed among the complications of bronchiectasis but it must be relatively rare. I have never seen a case and Perry and King[11] report but one case in a series of 400. *Metastatic abscesses,* usually in the brain but also in the liver and bones, are not uncommon.

Among our cases, three developed pneumonitis of such severity

and extent as to present physical and x-ray findings indistinguishable from lobar pneumonia. Eight additional cases had acute episodes of pneumonitis of milder degree. All of them recovered from the acute illness promptly. The temperature was usually normal by the end of the second or third day. However, rales in the lungs were persistent, and cough and expectoration were usually worse than before the acute illness. This chain of events, prompt recovery from an acute illness resembling pneumonia, but with persistence of rales, cough and expectoration is almost diagnostic of bronchiectasis, and it should lead to the making of bronchograms in every case.

One of our patients, with advanced bronchiectasis of the left lower lobe simultaneously developed osteomyelitis of the right 6th and 7th ribs and multiple frontal lobe cerebral abscesses. His death was the only one of this series.

TREATMENT

Prophylaxis. The essence of the prophylactic treatment of bronchiectasis is the constant awareness on the part of the attending physician of the possibility of the development of bronchiectasis in any child or young adult suffering from an acute respiratory infection. If purulent secretion develops during such illness, complete drainage of the bronchial tree must be insured. Medically this is done by altering the bronchial secretions and by the encouragement of gravity drainage of the bronchi.

Various methods are used to modify the sputum. Of the expectorants that are commonly used I have found potassium iodide to be the only one of any use. Steam inhalations may have some effect during the acute stage of bronchitis. Inhalations of carbon dioxide are said to thin bronchial secretions and by hyperventilating the respiratory tract tend to dislodge tenacious mucus. It is most useful in the presence of atelectasis.

Bronchiectasis occurs, with rare exceptions, in the most dependent bronchi. It is therefore obvious that one of the ways to discourage its development is to place the patient with an acute respiratory illness in a position such that these bronchi are no longer the most dependent. This is done very simply by having him lie in a prone position. The inclination of the trachea is dorsally, and with the patient in a prone position sputum is able to travel downhill all the way from the posterior bronchi in the bases of the lungs to the pharynx. The average patient, if left to his own devises, lies in bed on his back, usually propped up on pillows. In this position, gravity drainage of the posterior lower lobe bronchi is the most severely hampered.

In addition to instructing patients to maintain a prone position

as much as possible, I have made it a practice for years to use postural drainage, as it is used in cases of bronchiectasis, on all patients with purulent bronchial secretion; unless severe illness of the patient or cardiovascular conditions contraindicate it.

If, with the preceding regime, cough and expectoration do not promptly subside, bronchoscopic aspiration is indicated. In all cases in which cough, expectoration, rales in the bases of the lungs, and radiographic evidence of pulmonary infiltration or atelectasis persist for more than a few weeks after a respiratory infection, bronchograms should be made.

The modern chemotherapeutic agents deserve mention as factors in the prophylaxis of bronchiectasis. Inasmuch as streptococci are the chief offenders in this disease, the sulfonamides and penicillin should prove effective in many cases of respiratory infection in breaking the chain of events that leads to bronchiectasis before it has been completed. Nevertheless, it was strikingly apparent in this series that chemotherapy, particularly with the sulfonamides, does not provide certain insurance against the development of bronchiectasis, for many cases developed following sulfonamide therapy for the primary pneumonia. It is too early to evaluate penicillin in this regard.

Medical Treatment. In established bronchiectasis, medical treatment is palliative only. In none but the very earliest cases can cure by medical means be hoped for. Postural drainage is the only one of these measures that is of positive value and its value is distinctly limited. To get the greatest benefit from it, the patient's chest must be inverted and maintained in as nearly vertical a position as possible for as long as the patient can tolerate this position. This means that he must lie on the edge of something at least as high as the ordinary hospital bed, supported by his thighs on the bed or table and his hands on the floor. This position should be demonstrated to him to be sure that he understands it and he should be instructed to maintain it as long as possible, even though secretion is not immediately forthcoming. It is only from large saccular dilatations that the secretion will pour forth as from an inverted cup. With the chest inverted the difficulty in draining secretion from slightly or moderately dilated bronchi is the same as that encountered in draining liquid, especially if it is highly viscous, from any cylindrical tube closed at its upper end. Voluntary coughing, not too violently, is of considerable aid in dislodging the secretion. This drainage should be done at frequent intervals if maximum benefit is to be obtained. A good schedule is to do it upon arising, before each meal, and upon retiring. Postural drainage is, of course, contraindicated in debilitated patients and in those with hypertension or evidence of

arteriosclerosis. The increased hydrostatic pressure in the vessels of the head might easily cause retinal or cerebral hemorrhage in the presence of diseased arteries.

Surgical treatment. The establishment of lobectomy as the treatment of choice in bronchiectasis within the period of a little more than a decade is one of the more important recent developments in medicine. In their monumental volune *"Surgical Diseases of the Chest"* published in 1935, Graham, Singer, and Ballon[4] remark concerning this procedure, "....the operation remains even in the best hands one of the most serious procedures in the surgical repertory....a patient with bronchiectasis who submits to a lobectomy runs about a 15 to 20 per cent risk of dying because of the operation....and if he recovers from the operation he has only about a 65 per cent chance of having a thoroughly satisfactory result with solid healing of the wound."

This situation is entirely changed. In the best of hands, the mortality for lobectomy is now 4 per cent or less. With more precise bronchography a thoracic surgeon may now operate with more assurance that he is going to remove all of the diseased lobes, thus attain a higher percentage of complete cures. Refinements in surgical technique and the use of the sulfonamides and penicillin prophylactically against post-operative infections have reduced to an almost negligible figure the percentage of permanent bronchial fistulae and failures of wound healing. A physician who nowadays fails to refer a case of bronchiectasis suitable for lobectomy to a chest surgeon is almost as derelict in his duty as one who treats acute appendicitis with ice packs. As Churchill[3] has put it, "....the time has arrived when the medical profession can stop fumbling with bronchiectasis...."

Nevertheless, it was possible to do lobectomies in but 7 cases of this series, in spite of the high percentage of cases suitable for the operation. The biggest obstacle in our path was the refusal of patients to submit to the operation.

SUMMARY

1. Bronchiectasis is a common disease. Among 9754 cases admitted to a large naval hospital 59 cases were found, an incidence of 0.6 per cent. This incidence is comparable to that of pulmonary tuberculosis as determined by mass surveys.

2. In only six patients of this series had a diagnosis of bronchiectasis been made prior to their admission to the naval hospital from which final disposition was made.

3. Most cases of bronchiectasis follow streptococcal respiratory infection of one type or another, the most common being broncho-

pneumonia. Sinusitis was not found commonly to precede or accompany bronchiectasis.

4. The expectoration of large quantities of foul sputum and clubbing of the fingers were found to be distinctly uncommon in this series. Foul sputum was found in but two cases and clubbing of the fingers in but one.

5. Persistent cough and expectoration following a respiratory illness occurred in every case in this series. The persistence of this combination of symptoms following an acute respiratory illness should always make the physician think of bronchiectasis.

6. The commonest radiographic findings were thickening and fuzziness of the lung markings at the bases of the lungs. When these findings occur together with persistent cough and expectoration, bronchiectasis is found on making bronchograms, in a high percentage of cases.

7. Bronchograms made with iodized oil are the only sure means of diagnosing bronchiectasis. They are simply and easily made by means of the supraglottic method of instilling iodized oil into the tracheobronchial tree.

8. Lobectomy is as surely established as the treatment for certain types of bronchiectasis as is appendectomy for the treatment of acute appendicitis, but its usefulness has been sharply limited by the lateness with which most cases of bronchiectasis are diagnosed.

I wish to express my sincere appreciation to Dr. John C. Jones of Los Angeles for performing the lobectomies done in this series.

3616 Main Street.

RESUMEN

1. La bronquiectasia es una enfermedad común. Entre 9754 casos admitidos a un espacioso hospital naval se descubrieron 59 casos, o sea una frecuencia del 0.6 por ciento. Esta frecuencia es comparable con la de la tuberculosis pulmonar determinada por censos colectivos.

2. Solamente en seis pacientes de esta serie se había hecho un diagnóstico de bronquiectasia con anterioridad a su ingreso al hospital naval donde se llevó a cabo la disposición final del caso.

3. La mayor parte de los casos de bronquiectasia siguen a infecciones de las vías respiratorias por estreptococos, de un tipo u otro, el más común de los cuales es la bronconeumonía. No se encontró que la senositis precediera o acompañara comúnmente a la bronquiectasia.

4. La expectoración de grandes cantidades de esputo fétido y el ensanchamiento de la punta de los dedos fueron hallazgos muy raros en esta serie. Solamente se encontró esputo fétido en dos casos y ensanchamiento de la punta de los dedos en uno sólo.

5. La tos y la expectoración persistentes consecutivas a una afección de las vías respiratorias se presentó en todos los casos de esta serie. La persistencia de esta combinación de síntomas como consecuencia de una enfermedad aguda de las vías respiratorias, siempre debe hacer pensar al médico en la bronquiectasia.

6. Los hallazgos radiográficos más comunes fueron engrosamiento y obscurecimiento de las marcas pulmonares en las bases de los pulmones. Cuando estos hallazgos van acompañados de tos y expectoración persistentes, los broncogramas revelan bronquiectasia en un alto porcentaje de casos.

7. Los broncogramas con aceite yodado son el único método certero de diagnosticar la bronquiectasia. Se llevan a cabo estos broncogramas con sencillez y facilidad mediante el método supraglótico de introducir el aceite yodado en el árbol tráqueobronquial.

8. La lobectomía está tan firmemente establecida como tratamiento para ciertos tipos de bronquiectasia como lo está la apendectomía en el tratamiento de la apendicitis aguda; pero su utilidad ha sido sumamente limitada por lo tarde que se diagnostican la mayor parte de los casos de bronquiectasia.

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Proposed State Tuberculosis Control Program*

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With the appearance of favorable federal legislation and grants-in-aid, tuberculosis control activities are being stimulated throughout the entire nation. Educational campaigns and surveys carried on through many years in the past have made the public tuberculosis conscious. In order to take full advantage of these favorable conditions, it now becomes necessary to make an inventory of present and past State control procedures and to make such changes and additions as will insure a successful long-term program.

Any control program, if it is to fulfill its mission, must embody the basic principles of disease control—case-finding, case-isolation, and case-prevention. Since at this writing, no immunizing method has been proved to be of long-time value, the prevention of secondary cases depends upon finding the source cases early and treating them before they become infectious. Current treatment implies initial hospitalization in a sanatorium.

Tuberculosis is a chronic disease and there are no specific cures as yet. Under certain conditions, such as defined in the Diagnostic Standards and Classification of Tuberculosis, patients may be classified as apparently arrested, arrested, and even apparently cured. No cases are classified as cured. Obviously then, the occupation, and the home and working conditions of a person with inactive disease must be evaluated and altered, if necessary, to prevent the reactivation of the disease. Thus, home welfare and vocational rehabilitation, where required, along with case-finding, case-isolation, and physical restoration must be provided if we are to improve our methods in the control of tuberculosis.

It therefore follows that an adequate tuberculosis control program should embody the following four basic functions:

(1) Case-finding,
(2) Isolation (hospitalization and treatment),
(3) Rehabilitation and family welfare, and
(4) Prevention.

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Case-Finding

Experience has shown us that tuberculosis develops in the lungs long before any physical signs or symptoms appear. The vast majority of patients are not aware of their condition until symptoms develop or until a chance examination of their chest reveals the disease. In many instances, the disease develops and becomes arrested without the patient being aware of any disease at any time.

It naturally followed that case-finding programs became founded and developed on tuberculin tests and chest x-rays. The time and money factors involved in such programs were obstacles to expected accomplishments on a State-wide basis. We still find over 85 per cent of the patients admitted to the sanatoria in the advanced stage of the disease.

For many years, the Minnesota Department of Health has been concerned with the large number of cases which were first reported at the time of or just prior to death. In 1945, 15 per cent of the deaths from tuberculosis were first reported at the time of death. Even though this percentage is a marked decrease from 45 per cent in 1923, the percentage is still too large. Mortality statistics for the United States reveal that in 1944 thirty-six (36) per cent of the tuberculosis deaths occurred outside of institutions and hospitals. Only 36.6 per cent of the tuberculosis deaths for that year occurred in tuberculosis hospitals or nervous and mental institutions. These figures represent only a slight betterment over the 1939-1941 averages. The implications of serious intimate exposure to contacts by these open cases is evident.

Obviously, newer methods were necessary for mass examinations.

As a result of the work of deAbreu, Potter, Hilleboe, and others, it is now possible for us to employ mobile photofluorographic units capable of taking large numbers of chest films daily. With the use of such equipment mass radiography or photofluorography can be done at lower costs and with economy of personnel and time. It is to be expected that further experiences in the field will bring further improvements in our mass attack on tuberculosis. The use of the mobile x-ray unit for mass chest surveys should not cause the elimination of other satisfactory case-finding procedures.

The ideal case-finding method would be not only to tuberculin test all persons in the community with either 1:1000 dilution (0.1 mg.) of O.T. or the two test dose of P.P.D., but also to chest x-ray all the persons regardless of the tuberculin reaction every year.

Obviously, a program of this type would not be practical. A very

effective case-finding program can be carried out by applying these procedures on a need basis. Each area should plan its own program according to the local situation.

An effective case-finding program, then, should embrace these three activities:

(1) Mass photofluorography (to include chest x-ray examinations of hospital employees and patients),
(2) Tuberculin surveys, and
(3) Case-contact follow-up.

Mass Photofluorography

The details for the operation of the State owned units are recommended as follows:

1. *Personnel*

A. Fixed, on State payroll.

(1) Technician-driver, responsible for the operation and the maintenance of the unit in the field.
(2) Clerk-typist, responsible for the necessary unit clerical work, including completion of the required unit forms.
(3) Public Health Nurse, with special organizational ability, responsible for the advance organization and stimulation of the survey project; also responsible for public relations. She will act as "advance agent" for the unit and enter the community well in advance of the unit. It is not intended that the nurse remain with the unit during the entire period of the survey but will move on to the next project area as soon as practicable. It is understandable that in surveys of industries, schools, and other selected groups it will not be necessary for the nurse to remain any length of time, and that in other areas it may be necessary for her to remain a longer period of time.

B. Attached personnel, local.

It will be necessary to obtain from local sanatoria, and local voluntary and official public health and welfare agencies such additional personnel as will be required to fill out an efficient organization. These workers may either be paid or voluntary, but if paid, they will have to be paid from local resources. The amount and type of assistance required and obtained will vary in different communities.

2. *Planning the project*

A. Selection of the area for mass survey.

It is expected that the appearance of the mobile unit in the field will stimulate a great deal of interest by lay and medical groups for the use of the unit in their area. The function of the unit is to find tuberculosis cases. In view of the fact that certain population groups have a greater tuberculosis potential than others, the selection of areas for mass survey must be carefully considered. It is not necessary to make a survey area county-wide, but the population group chosen should be accessible and large enough to

make the project practicable. In some areas the project may be solely industrial.

Requests from all lay or professional groups will be accepted. All requests for surveys will be submitted to the State Department of Health. These requests will then be screened by the Tuberculosis Control Officer, State Department of Health; and the Chief, Medical Services Unit, Division of Social Welfare. Final selection of the area to be surveyed will be determined by the Tuberculosis Control Officer, State Department of Health; Chief, Medical Services Unit; and Director of local sanatorium or State Sanatorium concerned. The component medical society will be consulted prior to placing the survey area on the calendar. Priorities will be determined from study of the tuberculosis problem, local cooperation and public interest, available local personnel for follow-up, and such other local conditions as may influence the success or failure of the project.

It therefore follows that areas or population groups chosen for mass survey should be selected on the basis of need. These are the indices which may be used to determine the need of such surveys:

(1) Number of deaths from tuberculosis and its rate.
(2) Ratio of number of new cases discovered to number of annual deaths.
(3) Number of cases reported at time of death or shortly before death.
(4) Number of sanatorium cases.

A study of these indices will give us a measure of the tuberculosis problem in various areas. However, it is to be recognized that in analyzing deaths and death rates, conclusions are apt to be faulty because of a number of variables which are difficult to control.

Since certain population groups have a greater tuberculosis potential than others, it is advisable that initially the following groups be used as focal points for mass surveys.

(1) Known and suspected cases and their contacts.
(2) Employees in industries. Mass surveys in this group are best by separate industry.
(3) Employees of institutions and hospitals.
(4) Persons residing in low economic areas.
(5) Students in colleges and senior high schools.

B. Preparation for survey.

When the area or group for a mass survey has been selected, sufficient time should be allowed for preparation. The amount of time necessary will vary in different localities. Generally speaking, at least two months will be required in most places. However, it is understandable that in industries or in selected group surveys a great deal less time may be all that is necessary.

It is desirable that the local, district, or State Health Department sponsor the case-finding program. Every effort should be made to have the local or State Sanatorium participate actively in the program. The amount of sanatorium collaboration will depend upon the proximity of the sanatorium to the survey area and upon the active interest shown by the sanatorium director. The voluntary

Public Health Association (Christmas Seal Organization) and its local organizations should be invited to participate actively in the project. In counties not affiliated with the National Tuberculosis Association, the invitation should be rendered to the local independent association. Other official and voluntary health and welfare agencies should also be asked to participate.

The following procedure is recommended:

(1) Selection of the area to be surveyed in accordance with instructions in 2-A above. Consideration should be given all requests received by the State Department of Health, but need should govern selection of the area.

(2) Tuberculosis Control Officer should visit area selected and confer with Director of local sanatorium or State Sanatorium and determine with him degree of sanatorium collaboration. Conferences should then be held with the appropriate committee of the component medical society and then with the official and voluntary health and welfare agents and agencies. A general advisory committee will then be formed. The committee should consist of representatives of the medical profession, religious, fraternal and farm groups, management and labor, and civil and educational leaders. The exact make-up of the committee will vary in different communities in accordance with local conditions.

(3) Well in advance of the date of the survey the public health nurse of the mobile unit to be sent to the area should enter the community and with a working sub-committee of the general advisory committee, organize the campaign. The organization should include the formation of groups of voluntary workers, each group of which should be given definite duties. The planning should include the following:

(a) Intensive educational campaign consisting of speakers, posters, radio and newspaper notices, pamphlets, films, etc. The educational campaign should be under the supervision of the Educational Service Unit, State Department of Health, in collaboration with the voluntary Public Health Association.

(b) Arrangements for the inclusion of local industries and schools in the survey.

(c) Procedure for visiting homes in which there are cases and contacts.

(d) Arrangements for an intensive drive to get in as many of the general public as possible, emphasis placed on barbers, beauty operators, food handlers, bartenders, and hotel and laundry employees.

The entire preparatory planning and work should be so arranged that the unit will arrive at the peak of the stimulation so that it can be kept busy during the period of its stay there.

(4) Before the program is started in a community, arrangements must be made with the appropriate authorities for hospitalization of cases found and in need of treatment.

C. Role to be played by county sanatoria. This should be integrated with the Tuberculosis Committee of the State Medical Society.

3. Operation of units.

A. Miniature film, reading of film.

All examinees except cases should report to the unit in accordance with their appointment time. They will each report to the unit clerk and receive from her an identification card (mass radiography form No. 1), which they will complete if not previously filled out, and then give this form to the technician as they report to him for their x-ray.

Each evening, project films exposed during the day should be processed in the unit and then promptly sent to the place where the films are to be reviewed by the central office. In some localities, it may be necessary for the roentgenologist of the State Department of Health to visit the unit and read the films while there.

The report of the project films should be sent without delay to the unit clerk. If the report returned is essentially negative, then the clerk will give the properly filled out tabs from mass radiography form No. 1 to the physician designated by the patient. The examinees will be notified to report to their physicians for the findings. If a retake on 14" x 17" celluloid film is indicated, the examinee will be notified by the unit clerk that a re-examination is necessary because the project film was found to be unsatisfactory.

B. 14" x 17" celluloid films.

Prior to the entrance of the mobile unit in the area, it should be determined by conference with the component medical society as to whether the x-ray examinations of cases and suspects and retakes of project film on 14" x 17" celluloid films should be done by the mobile unit or through local facilities. The unit is prepared to take 14" x 17" roentgenograms if such are desirable. The procedure should be followed whether the abnormal project film findings reveal suspected tuberculous or non-tuberculous lesions.

If the 14" x 17" films are taken by the mobile unit, they should be processed either at the nearest local hospital or sanatorium or sent unprocessed to the central office where they are processed, if necessary, and interpreted by the state roentgenologist. If the 14" x 17" films are taken by the local physician, clinic or hospital, then the local physicians should be urged to submit these films to the central office for interpretation.

All findings should be sent to the designated private physician or clinic or hospital and copies submitted to the Director of the local sanatorium (or State Sanatorium), and the District Health Unit or County Public Health Nurse.

4. *Follow-up.*

Following the operation of the mobile unit in the area it is necessary that immediate follow-up be made of the cases found through the survey, and of the cases and suspects and their contacts who have not reported for their examinations. The follow-up is an essential part of the program and should be done without delay in order to determine those who are in need of active treatment or observation. Appropriate disposition should be made of all cases or suspects as early as possible.

Tuberculin surveys:

Tuberculin surveys when properly conducted are necessary for a well-balanced control program. Their use is recommended as follows:

(1) to determine the rate of infection in a community,
(2) for epidemiological investigation,
(3) for case-finding in a low tuberculosis area.

If a tuberculin testing program is planned, those groups and ages should be tested which will accomplish the purpose intended. In all events, positive reactors should be used as a lead in tracing cases back to the homes, shops, and/or schools. If this is not done, then the real value of such programs is reduced.

If a large enough proportion of the general public is x-rayed at accepted intervals, most of the infectious active cases of tuberculosis will be discovered and removed from the community. Since, at the present time, there are an insufficient number of mobile units and personnel to operate these units, tuberculin surveys can be effectively employed in the interim. It is hoped that analysis of the x-ray surveys now being conducted will determine the optimum time for re-ray of a community.

Contact follow-up:

It has been established that the chances of developing reinfection tuberculosis is directly proportional to the degree and amount of exposure to an infectious case. For that reason, intimate contacts of active cases will require closer medical supervision than casual contacts or positive reactors from unknown sources. It has also been established that the vast majority of contacts over twelve years of age who subsequently develop tuberculosis do so within three years after exposure.

It therefore follows that all contacts should be closely supervised during the exposure period and for three years after the exposure has been interrupted. The interval for re-examination should be determined by the attending physician. However, after the three-year period, annual chest x-ray should suffice. Here again, the attending physician, because of previous findings or knowledge of the patient's home conditions, may desire to continue the contact on closer medical supervision.

In addition, mass x-ray and tuberculin surveys will uncover a significant number of cases which will have to be studied clinically for diagnosis and activity.

The importance of the private physician should not be overlooked. He is the one who can find a large number of cases in the important population group not entirely reached by other procedures. He should be encouraged to employ a more aggressive anti-

tuberculosis action. In addition, he is consulted by the confirmed case as to treatment, etc.

At the last State Medical Association Annual Meeting, the Council and House of Delegates of the Minnesota State Medical Association approved the report by their Committee on Tuberculosis, in which was included the recommendation that medical and sanatorium care should be arranged for any active case of tuberculosis.

Isolation (Hospitalization and Treatment)

Isolation is an important procedure in our control program. The earlier a case is isolated the less probability the contacts will develop tuberculosis. At present over 85 per cent of sanatorium admissions have advanced disease on admission. This alone would indicate that a large number of persons are being needlessly exposed to tubercle bacilli in their homes, shops, schools, etc.

Minnesota has 14 county sanatoria serving 44 counties and one State Sanatorium serving the remaining 43 counties. The combined bed capacity of the 15 sanatoria is 2014 beds. In 1944 there were a total of 699 deaths. If we exclude the 165 deaths which occurred in the State mental hospitals, prisons and veterans hospital, there were 3.9 beds per annual death. This is well above the standard of 2.5 beds per annual death. Thus, at first notice, it would seem as though there were sufficient beds to take care of our case load. It is to be expected that with the use of the mobile x-ray units for case-finding the active case load in this State will be greatly increased. The total Minnesota experiences to date indicate that about 1.5 per cent of apparently healthy persons given x-ray examinations show significant tuberculosis shadows. However, about 0.1 per cent of all persons examined will require sanatorium care for observation or treatment.

If the 0.1 per cent rate continues in the rest of the State it is to be expected that instead of having bed vacancies in the sanatoria, present case-finding methods will uncover more cases than there are bed vacancies. It may be necessary in order to utilize all available beds that some patients unable to obtain hospitalization in their own area be hospitalized in an adjacent sanatoria for such temporary hospitalization. Fortunately, the large majority of cases found on x-ray surveys are in the early stage and should require only a short stay in the sanatorium. It is therefore estimated that the over-load of patients in the sanatoria will be only of a short duration, perhaps five years following a survey, after which time vacancies created in sanatoria will be of a more permanent nature. Perhaps for this period one may estimate the bed requirements at five beds per annual death.

To the question as to whether certain small sanatoria should

be closed, we believe the answer is to wait and see what the surveys will produce. It is no doubt true that the smaller sanatoria do not have all the facilities required for adequate surgical care of the tuberculous. Perhaps, in order to make such treatment immediately available to all Minnesota residents, it might be well to use the smaller sanatoria for the chronic ambulatory and rehabilitation cases and the larger sanatoria for the patients in need of a complete treatment center. At any rate, an effective tuberculosis program calls for the immediate hospitalization of all infectious cases (actual and potential). That is basic.

Rehabilitation

Because of the chronic nature of the disease, many patients are required to remain in a sanatorium for long periods of time and are physically incapable of full time employment upon their discharge from the sanatorium. Because of economic necessity, many of them are compelled to return to their employment earlier than is desirable or to employment which is detrimental to their future health.

The purpose of rehabilitation is two-fold; first, to return to the patient his self-respect by directing him into avenues of work in which he is not discriminated against nor vocationally handicapped and second, to return to him his economic independence as rapidly as his health permits.

The rehabilitation program should consist of the following functions:

(1) Mental restoration,
(2) Physical restoration,
(3) Vocational and medical guidance,
(4) Training,
(5) Placement.

Methods chosen to operate the rehabilitation program must be combined into an orderly process. It is necessary at all times to select carefully the patients eligible for rehabilitation care. Under no circumstances should anyone be compelled to accept rehabilitation.

The rehabilitation program in Minnesota is a function of the State Department of Education. Seventy persons, or 14.5 per cent of the total disabled persons rehabilitated in 1945 were former tuberculous patients. It is hoped that this service will reach more persons eligible for it. On July 6, 1943 Congress passed the Borden-LaFollette Act. This legislation made possible vocational rehabilitation to all disabled persons who can profit from this service and broadened the scope of the service under Federal-State program.

Voluntary health and welfare agencies can assist in the rehabilitation program in four ways:

(1) Provide teachers and occupational therapists to the smaller sanatoria.

(2) Provide supplemental financial and material assistance to dependents of patients.

(3) Educate management and general public not to discriminate against the employment of selected ex-patients.

(4) Provide supplemental financial or material assistance to trainees either as grants or loans.

Prevention

In the foregoing discussion under case-finding, hospitalization and treatment, and rehabilitation, one can readily recognize the value of early case-finding, early isolation, and adequate rehabilitation as potent factors in the prevention of the spread of tuberculosis.

The following additional remarks are worthy of emphasis at the risk of repetition:

(1) The value to the community of isolating open cases is so great that it should be incumbent upon them to provide free sanatorium care for all their residents with active tuberculosis and to those non-residents who are found to be infectious. The cooperation of the attending physician, the consultant, the health officer and the community is necessary.

(2) Once a patient has been admitted to the sanatorium, he should remain there until discharged by the Superintendent as having received maximum hospital care and being non-infectious.

(3) Follow-up examinations of post-sanatorium cases or observation cases should always include submission of sputa specimens to the State Department of Health Laboratories.

(4) Periodic chest examinations (or tuberculin tests) of teachers, school employees and contacts of cases. Teachers should be required to have their examinations at time of employment and at least every two years thereafter.

(5) The annual chest x-ray examination of barbers, beauty operators, food handlers, bartenders and the like would assist in locating active tuberculosis in persons coming in close association with the general public. The value of this procedure in tourist or trade centers can not be overstated.

(6) Improvement of nutrition and housing.

In this proposed expanded tuberculosis control program with the use of mobile x-ray units, it is expected that the combined coordinated attack on tuberculosis by the State Department of Health, sanatoria, and other official and voluntary health and welfare agencies will produce results. It is not the desire of the State Department of Health to interfere nor to hinder the good work of the sanatoria and other agencies which has been so fruitful in the past, but rather to lend them assistance and the authority which is at our means to provide.

The Medical Management of the Bronchiectatic Patient*

ELLIOTT MENDENHALL, M.D., F.C.C.P.
Dallas, Texas

The clinical entity known as bronchiectasis is being recognized a great deal more than formerly. This disease was first described by Laennec in 1819, and received a good deal of study by the pathologists. Since there was no satisfactory treatment, the practitioners gave little attention to it until Killian introduced the bronchoscope in 1880. Stimulated by Jackson, further study of the bronchial tree has added to our knowledge of bronchiectasis; and this study and improved methods of diagnosis are partly responsible for the increased reports on this disease.

During the past decade the surgical treatment of bronchiectasis has gained favor. Unfortunately only about one-half the cases are suitable for surgical treatment. This leaves a vast number of individuals who are still wholly dependent on medical treatment for such relief as they can get. Because of the physical, social, and economic handicaps of this crippling disease, these people are entitled to as much alleviation as can be given them by medical management.

When trying to establish or to evaluate a treatment, it is well to keep in mind the pathology and the pathologic physiology which one is trying to correct. There have been expressed several theories of the etiology of bronchiectasis. After all has been said, however, it seems to me that the essential elements are bronchial obstruction and infection. To me, this *is* bronchiectasis. In the medical treatment of this condition the aim is first to relieve the obstruction as much as possible, thereby allowing re-aeration of the infected lung tissue. This in itself aids in the second objective, that is, to inhibit the growth of the pathogenic bacterial flora, thus relieving to some extent the unpleasant symptoms due to these organisms.

Prevention

The preventive treatment of bronchiectasis is largely the responsibility of the pediatrician. All respiratory diseases in children should be treated most carefully and thoroughly. Convalescence should be prolonged whenever necessary to restore the child to

*Presented before the Texas Chapter, American College of Chest Physicians, Galveston, Texas, May 6, 1946.

good general health and to re-aerate all parts of the lungs. Foreign bodies in the bronchi should be removed immediately to prevent secondary changes at their point of lodgment and beyond. Acute infections in the bronchial tree and lungs of adults should receive the same careful attention, especially influenza, bronchial pneumonia, and atypical pneumonia. Postoperative atelectasis should be relieved immediately by bronchoscopy if necessary.

General Measures

It is axiomatic to state that bronchiectatic patients should be kept in the best possible general health. Foci of infection, particularly about the mouth and upper respiratory tract should be removed when possible. Adequate nutrition, including a sufficient supply of vitamins and minerals, is important. These patients, as far as is humanly possible, should avoid exposure to respiratory infections and exposure to inclement weather. Where it is economically possible, it is often helpful for them to live in an equable climate, at different locations at different seasons if necessary. The use of autogenous vaccines is permissible, though there is nothing specific in their action, nor do they give spectacular results. I have used them for years. They have never harmed a patient, and in many instances, patients have had fewer acute respiratory episodes during the winter and spring months, and in many instances the bronchiectatic sputum has decreased. I never promise a patient any favorable results from their use, and I do not insist on their use if a patient objects. This, I deem a wise and honest precaution.

The administration of expectorant cough mixtures, iodides, creosote, and the hypodermic injection of some form of iodide should be tried. These drugs help to deoderize and to thin the secretions, thereby facilitating drainage. When there is not sufficient natural sunshine, the use of ultraviolet light is permissable. The administration of sulfonamides by mouth and penicillin intramuscularly should be used in selected cases. Penicillin can be given once daily, 300,000 units at a dose, until two or three million units have been given. This can be repeated as indicated. Toxic effects are unusual and mild. When fusiform bacilli and spirochoetes are numerous in the sputum, neoarsphenamine may be given intravenously, but in my experience this has not proved of any decided benefit. Adrenalin sometimes relaxes the bronchi enough to help promote drainage.

Local Measures

Unless there is some distinct contra-indication, every bronchiectatic patient should have at least one bronchoscopic examina-

tion and drainage. This often relieves the bronchial obstruction by the removal of polyps, tenacious secretion or plugs of debris, or simply by dilatation of the bronchus. Postural drainage arranged to suit each individual is of prime importance. This is best done by having the patient lie on a cot, the foot of which is elevated to about a forty-five degree angle. He is instructed to lie five to ten minutes each on his back, on his abdomen, and on each side. He should cough and breathe deeply several times in each position. For drainage of the bronchi in the upper lobes the patient's chest is propped up, and he turns from side to side while coughing and breathing deeply.

The instillation into the bronchial tree of iodized oil, by its mechanical action, floats out a great deal of secretion. It may have a slight antiseptic action on the lining of the bronchi; although sufficient iodine is not liberated to be of much benefit in this fashion. I doubt that oft repeated instillations are of benefit, and may cause harm. The same is true of bronchial lavage. The use of antiseptic solutions directly in the bronchial tree is of doubtful value, and often produces bronchitis and pneumonitis. The use of steam inhalations with some volatile medicament, such as tincture of benzoin, relieves acute bronchial irritation. Vaporized penicillin offers some hope of temporary abatement of the infectious process. The success of this treatment depends on the content of the bronchial flora, on its proper use by the patient, and on his persistence. An oxygen tank with a regulator is attached to a special nebulizer. The patient places the mouth piece of the nebulizer well back in his mouth and sucks in a long breath as the oxygen flows over the solution of penicillin in the nebulizer. This carries the fine vapor into the bronchi. More penicillin will be retained if the patient holds his breath for a few seconds after each inhalation. I start my patients with a solution containing 10,000 units to the cubic centimeter of normal salt solution. If there is no evidence of bronchial irritation this is rapidly increased to 30,000 or 40,000 units per cubic centimeter. In this way, the patient can take from 100,000 to 200,000 units a day. I have used this treatment for over a year on patients without any untoward affects. It gives only temporary relief, but it can be repeated without harm. If the oxygen apparatus is not available, the patient can use the hand bulb that comes with the nebulizer, though that is not quite as satisfactory.

It is realized that none of the above measures offer any specific remedy or cure for bronchiectasis, but some of them will bring some measure of relief to an otherwise miserable patient. They should be tried in those cases not suitable for surgery.

SUMMARY

More cases of bronchiectasis are being recognized. About one-half of the patients are not suitable for surgical treatment, but they deserve as much relief as medical care can offer. Relief of bronchial obstruction, promotion of drainage, and inhibition of the infectious process offer some hope of temporary restoration of physiologic function. We should strive by these means to give an otherwise sick and miserable patient some measure of relief and help restore him to his rightful place in society.

RESUMEN

Se están descubriendo más casos de bronquiectasia. El tratamiento quirúrgico no es aplicable a la mitad de los enfermos, más o menos, pero ellos merecen tanto alivio como pueda ofrecerles la atención médica. El tratamiento de la obstrucción bronquial, el establecimiento de drenaje y la inhibición del proceso infeccioso ofrecen alguna esperanza de la restauración temporaria de la función fisiológica. En esta forma debemos esforzarnos por proporcionarle al paciente, de otro modo enfermo y en un estado miserable, alguna medida de alivio, y a ayudar a restaurarlo a su puesto legítimo en la sociedad.

Subdiaphragmatic Aberrant Pulmonary Tissue*
(Case Report)

ANIBAL ROBERTO VALLE, M.D., F.C.C.P.
M. LAWRENCE WHITE JR., M.D.
Charlottesville, Virginia

The feasibility and popularity of exploratory thoracotomy for thoracic lesions has led to the discovery of congenital anomalies previously diagnosed only at the autopsy table. We present an unusual congenital malformation — accessory pulmonary tissue contained within the sac of a diaphragmatic hernia.

CASE REPORT

E. S., Case number 213013, a colored male infant nine months of age, was admitted to the University of Virginia Hospital on April 28, 1945. The mother's pregnancy and delivery were apparently normal. One previous pregnancy ended in stillbirth, another infant died of convulsions early in life and a third child at present is in good health. The mother stated that the patient had vomited "almost every feeding" since birth. The vomiting as described was projectile in nature and took place almost immediately after feeding. The vomitus consisted of undigested food without blood or mucus. No choking, coughing or cyanosis accompanied the vomiting. The bowel habits were normal and the stools, although small in amount, were normal in appearance. Obviously the child had failed to gain weight normally but had remained mentally alert.

Treatment prior to six weeks before admission had consisted of "drops" (? Atropine) taken by mouth. Failure to respond to this treatment prompted admission to a hospital elsewhere where after six weeks of medications the symptoms persisted. A diagnosis of diaphragmatic hernia was suspected and admission to the University of Virginia Hospital was advised.

Physical Examination: The general appearance of the patient was that of a poorly developed and nourished child in no acute distress. The weight was ten pounds, ten ounces, and the temperature was normal. The abdomen was distended and tympanitic, but no masses were palpated. Both testes were undescended, and phimosis was present. The remainder of the physical examination was essentially negative.

Laboratory Studies: Routine urine and stool examinations were negative. The hemoglobin was 13 grams (84.4 per cent), the erythrocyte count 4.96 million, and the leucocyte count 5,800 with 23 per cent segmented polymorphonuclear cells, 69 per cent lymphocytes, and 8 per cent monocytes. The Wassermann and tuberculin tests were negative.

Roentgen Studies: On April 30th barium studies of the gastro-intestinal tract were made. The esophagus was found to be shorter than

*From the Department of Surgery and Gynecology, University of Virginia School of Medicine, and University Hospital, Charlottesville, Virginia.

normal and moderately dilated. The barium passed quickly into a very large gas-filled stomach. The cardiac end of the stomach protruded through a diaphragmatic defect about two centimeters in diameter and formed an apparent herniation near the esophageal hiatus (Fig. 1). The barium readily passed through the pylorus and hourly films revealed a normal passage through the intestinal tract. The lung fields were clear.

The diagnosis of diaphragmatic hernia seemed confirmed by the roentgen studies but the constantly large and tympanitic stomach seemed to indicate an increase in vagal tonus. On this assumption, atropine sulfate (five minims three times daily) was administered for fifteen days

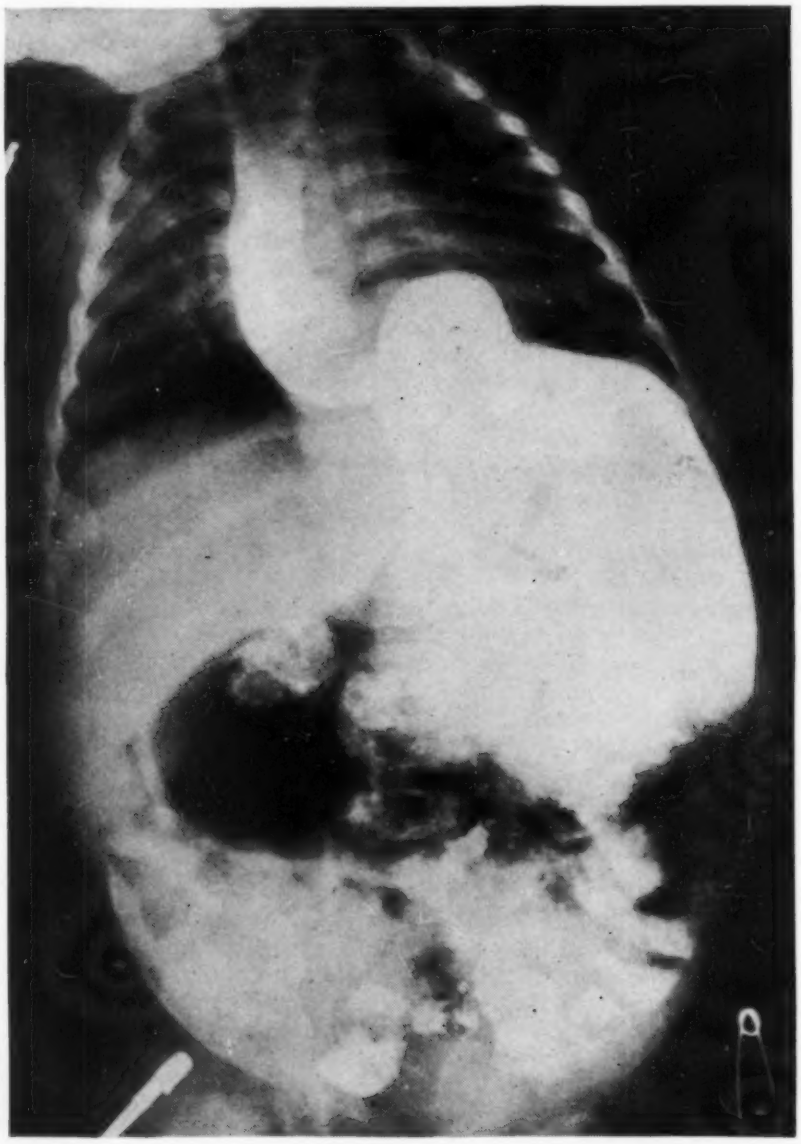

Figure 1: Roentgenogram after ingestion of barium shows the moderate dilatation of the esophagus, the marked distention of the stomach and the herniation of the stomach through the diaphragm.

and reexamination of the stomach by x-ray revealed some diminution in the size but the abdomen was still distended and tympanitic. There had been no appreciable gain in weight. Exploratory thoracotomy with repair of the diaphragmatic defect seemed imperative.

Operation: On May 24th the left hemithorax was explored by one of us (A.R.V.). A low postero-lateral incision was employed and the 8th rib resected subperiosteally from the transverse process to the cartilage. Upon opening the pleural cavity an obvious diaphragmatic herniation was seen posteriorly. There were no adhesions and the lower lobe of the lung was retracted easily to give excellent exposure of the whole diaphragm. The herniated mass was about the size of a hen's egg and was covered with thin tendinous diaphragm. By palpation it contained a solid viscus which could not be reduced. The thin diaphragm over the apex of the mass was divided and a dark red, friable mass of tissue presented. It was assumed at first that the spleen had become partially strangulated in the sac. With great difficulty the hernia sac was dissected from the mass. The dome of the diaphragm was divided radially in an anterior direction from the sac in order to better identify the subdiaphragmatic organs and to aid in the dissection of the contents of the hernia sac. The stomach, liver, spleen, and left kidney were readily identified and were normal on gross examination. A small portion of the stomach could at times have entered the sac beside the solid mass. This would explain the x-ray appearances. The mass contained in the hernia sac was considered to be an ectopic or accessory organ, probably kidney or liver from its appearance. With great difficulty it was dissected from the diaphragm and the posterior parietal peritoneum. Num-

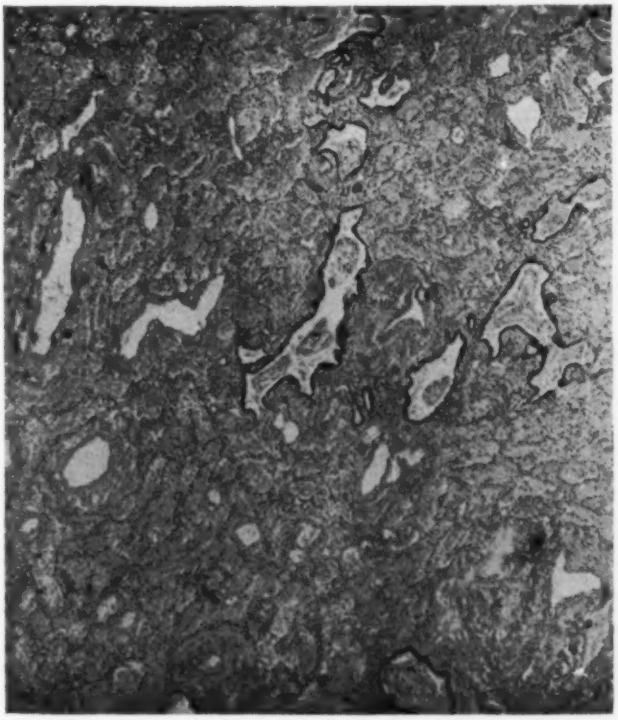

Figure 2: Photomicrograph of specimen showing pulmonary tissue with blood in the alveoli.

erous large vessels entered the mass, apparently from the aorta. One finger-sized projection was intimately attached to the esophagus but did not enter the lumen thereof. After ligation of the vessels, the mass was completely mobilized and removed. The diaphragmatic defect was then closed with interrupted sutures of fine silk. As the chest wall was being closed, the child's condition, which had been fairly good throughout, became suddenly worse and he died. The cause of death was probably operative shock.

Postmortem Examination: Examination of the body shortly after death revealed no significant abnormalities other than those resulting from the recent operation. The right kidney was small, weighing only ten grams as compared with the left which weighed twenty-five grams. No other structural defects were noted in either kidney. No other congenital anomalies were present. There was revealed no explanation of the persistently distended stomach.

The resected specimen measured 4 x 3 x 2 centimeters. On section "it was found to be dark reddish-brown throughout except for the center which was cystic and had a bloody-like material in it." The cystic cavity measured from one-half to one centimeter in diameter. Microscopically the specimen represented lung tissue, markedly congested with all of the alveoli filled with red blood cells (Fig. 2). There was no evidence of pneumonia or neoplasm.

DISCUSSION

A review of the literature revealed reference to two types of accessory or aberrant lung.[3] One occurs in the upper chest, the so-called tracheal lobe, and is connected by a bronchus to the trachea. The other, the so-called lower accessory lung, is found in the lower chest or upper abdomen and consists of sequestrated pulmonary tissue which has no connection with the bronchial tree. Of the latter type thirty-seven cases, including ours, have been recorded.

Although most cases reported were in stillborn infants the presence of this anomaly alone is not incompatible with life. Davies and Gunz[2] mention von Meyenburg's (1914) case in a six year old child, Rektorzik's case (1861) in an eighteen year old girl, and Springer's case (1898) in a forty-nine year old woman. As can be seen from Table I the anomaly occurs most frequently on the left side, only four cases of the thirty-seven being on the right. Diaphragmatic herniae are not infrequently associated with lower aberrant or accessory lungs, occurring in eleven (29.7 per cent) of the cases. The associated diaphragmatic herniae could be due to a hindrance to the closure of the diaphragm by the presence of this additional lung tissue extending downward toward the coelomic cavity.

There are many theories about the formation of this anomaly. However, we will briefly mention two which seem most plausible. The first theory, propounded by Cockayne and Gladstone,[1] involves the principle of sequestration. They state that at certain

embryonic stages, adhesions of lung mesenchyma to the coelomic mesothelium or to structures covered by this tissue are rather frequent. Whether this adherence is due to lack of separation of the lung mesenchyma from the mesenteric tissue, or to fusion of the two tissues, is undecided. However, these adhesions cause the lung mesenchyma to be displaced downward along with the foregut. This could plausibly account for the occurrence of diaphragmatic hernia and also for the attachment of the accessory lung to the lower esophagus and stomach, a finding in our case.

The second theory is based on the possibility of the development of a second primitive respiratory tract from the digestive tract.[3] This could be accomplished by the downward displacement of cells from the tracheoesophageal ridge with the growing foregut. A fact favoring this theory is that in almost every case the blood supply is derived from the aorta or the adrenal arteries. Venous drainage is usually through the azygos or hemiazygos veins.

It has been suggested that the occurrence of this anomaly on the left side is a consequence of the asymmetry that usually occurs in the early human embryo.[2]

From Table I it is seen that the present case is the sixth reported occurring beneath the diaphragm; and is the second reported in this location associated with a diaphragmatic hernia.

Thirty-six of the thirty-seven cases were discovered at autopsy. The present case appears to be the first diagnosed during life by an exploratory operation.

TABLE I

REPORTED CASES OF LOWER ACCESSORY LUNG
(including present case)

Author	*Side*		*Diaphragm*		*Diaphragmatic Hernia*
	Right	*Left*	*Above*	*Below*	
Beneke	..	+	..	+	+
Bert and Fischer	..	+	+	..	..
Carter and Osborn	..	+	+	..	..
Cockayne and Gladstone	..	+	+	..	+
Davies and Gunz	..	+	+	..	+
Davies and Gunz	..	+	+	..	..
Duerck	+	..	+	..	..
Freedlander and Gebauer	..	+	+	..	..
Gruber	..	+	+	..	+
Hückel	..	+	+	..	..
Humphry	..	+	+	..	..

Author	Side		Diaphragm		Diaphragmatic Hernia
	Right	Left	Above	Below	
Iwanoff	..	+	+	..	+
Kaplan	..	+	+	..	..
Kaup I	..	+	+	..	+
Kaup II	..	+	+	..	+
Kohn	..	+	+	..	+
Lewisohn	..	+	+	..	..
Morelli	+	..	+	..	+
Muller	..	+	+	..	..
Muus	..	+	+	..	..
Nordmann	..	+	+	..	+
Pineda	+	..	+	..	..
Quenzel	..	+	+	..	..
Rektorzik	..	+	+	..	..
Robsmann	..	+	..	+	..
Rokitansky	..	+	+	..	..
Ruge	..	+	+	..	..
Sachs	..	+	+	..	..
Scheidegger	..	+	+	..	..
Seltsam	..	+	..	+	..
Simpson	+	..	+	..	..
Springer	..	+	+	..	..
Valle and White	..	+	..	+	+
Vogel I	..	+	..	+	..
Vogel II	..	+	..	+	..
von Meyenburg	..	+	+	..	..
Wechsberg	..	+	+	..	..

*Gruenfeld and Gray[3] cited all of the above cases except those of Iwanoff, Morelli, and Nordmann, whose cases were cited by Davies and Gunz[2] in the presentation of their own patients.

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2 Davies, D. V., and Gunz, F. W.: "Two Cases of Lower Accessory Lung in the Human Subject," *J. Path. and Bact.*, 56: 417, 1944.
3 Gruenfeld, G. E., and Gray, S. H.: "Malformations of the Lung," *Arch. Path.*, 31: 392, 1941.

EDITORIAL

THE COLLEGE RESEARCH PROGRAM

Every Fellow, Associate Fellow and member of the College of Chest Physicians by reason of his expert information owes the world not only his routine services, but also any additional knowledge he is capable of contributing. Among our 2,000 members are many now engaged in research in various diseases of the chest and closely related subjects. Some are receiving support from institutions and research foundations, while others are personally financing their projects. Probably there are few, if any, who do not have research problems in progress or in mind. All could profit from and contribute to advancement of knowledge through a research organization within the College.

The assemblage of all available knowledge on any given subject is exceedingly important and often requires extensive research of the literature. However, unless this is done, scientific and clinical investigations are likely to be repeated or instituted with the lack of important facts already established. There are numerous subjects in the field of chest diseases for which all available information should be assembled. Thereafter the gaps in our knowledge should be filled in through scientific and clinical investigation.

The scope of the College is worldwide; therefore, in its research activities consideration must be given to each disease and condition that attacks the human chest. Every effort should be made to avoid duplication of research work already in progress by other organizations. The field is so large and the number of qualified workers is so small that duplication of effort among organizations would only postpone the discovery of much-needed information.

Dr. Charles M. Hendricks, a founder and now President of the College, has long appreciated the importance and, in fact, the necessity of organized research in chest disease. He has now appointed a Research Council, whose duty it will be to investigate various research projects that may be presented for consideration. Those that are regarded worthy will be given all possible support, including financial aid as funds permit.

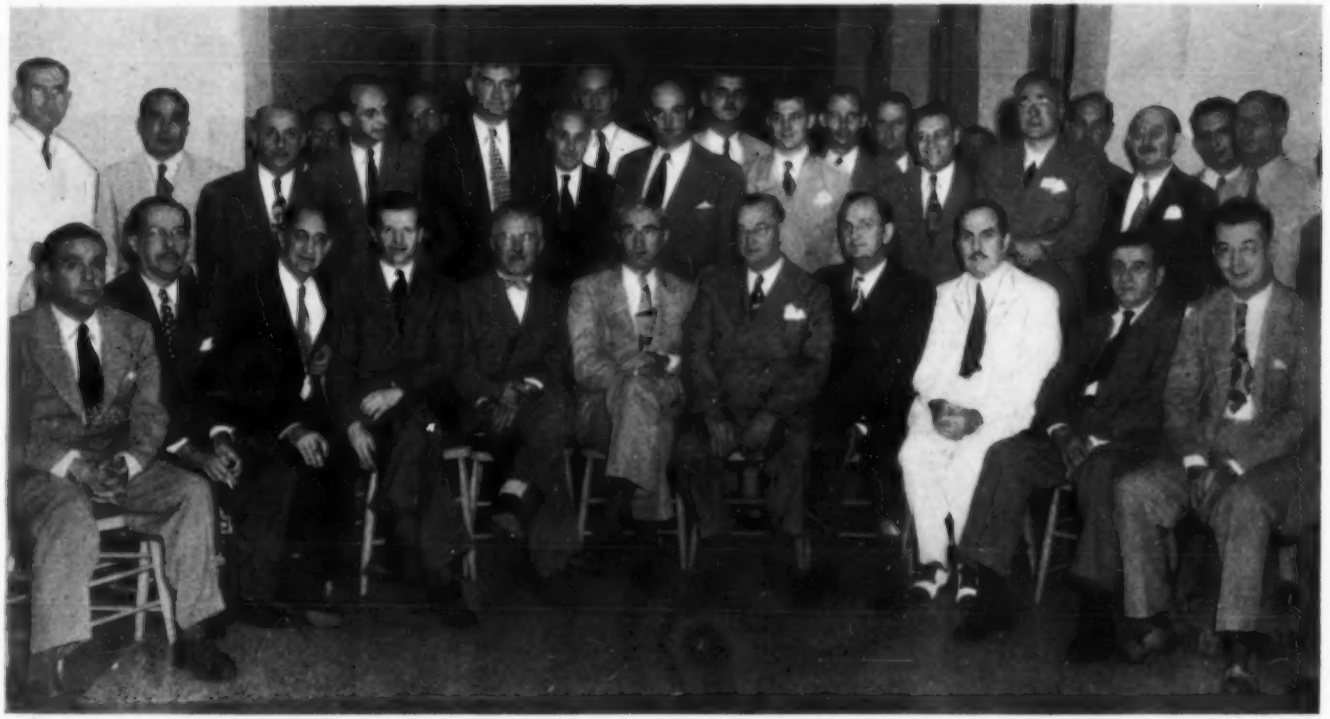
A sizable research fund will be made available. Already, through the efforts of Dr. Hendricks several College Fellows have subscribed $1,000 each and a number of others have expressed their intention to contribute this amount. In the near future a campaign will be launched, inviting every Fellow, Associate Fellow and member of the College to have a part in financing this important work. The immediate goal is $100,000, which should be readily forthcoming from the membership. Our annual dues are ridiculously low when one considers the benefits that accrue to each member. So much has been received for so little financial outlay that we can now take advantage of the opportunity to contribute to a fund which will perpetuate the good name and work of the College and promote the control of diseases of the chest throughout the world. With $100,000 as the initial fund much larger sums should be procurable from other sources.

The College must contribute knowledge pertaining to diseases of the chest if it is to continue to improve its already high standing among international medical organizations. Knowledge can be contributed only through research which, in turn, can be executed only through adequate funds. Therefore, the Research Council must succeed.

J.A.M.

Cuban Chapter Meeting, Havana, November 5, 1946, Mercedes Hospital

Officials of the Cuban Chapter and visiting College Fellows from the United States of America



Seated, left to right: Dr. Roberto Machado, Dr. Rene G. Mendoza, Dr. Paul A. Turner, Dr. David Waterman, Dr. Louis Mark, Dr. Carl C. Aven, Dr. J. Winthrop Peabody, Dr. Edward W. Hayes, Dr. Octavio Rivero, Dr. Minas Joannides, Dr. Herbert L. Mantz. Standing: Dr. Antonio Rodriguez Diaz, Dr. Ricardo Riera, Dr. I. L. Robbins, unidentified, Dr. Chevalier L. Jackson, Dr. Milton S. Lloyd, Dr. Benjamin L. Brock, Dr. William A. Hudson, Dr. Alvis E. Greer, Dr. Osler Abbott, unidentified, Dr. Sydney Jacobs, Dr. Duane Carr, Dr. William E. Denman, Mr. Murray Kornfeld, unidentified, Dr. John Roberts Phillips, Dr. Arnaldo Coro, Dr. A. J. Steiner, unidentified, Dr. Ricardo Sanchez Acosta.

College Chapter News

CUBAN CHAPTER

On Tuesday, November 5, the day after the meeting of the Southern Chapter of the College at Miami, a number of College officials, and their wives and other members of their families, journeyed to Havana to attend a meeting of the Cuban Chapter of the College. Following is a list of those who made the trip to Havana.

Dr. Osler Abbott, Emery, Georgia
Dr. and Mrs. Carl C. Aven, Atlanta, Georgia
Dr. Benjamin L. Brock, Oteen, North Carolina
Dr. Duane Carr, Memphis, Tennessee
Dr. William E. Denman, Memphis, Tennessee
Dr. and Mrs. Alvis E. Greer, Houston, Texas
Dr. and Mrs. Edward W. Hayes, Monrovia, California
Dr. and Mrs. William A. Hudson, Detroit, Michigan
Dr. and Mrs. Chevalier L. Jackson, Philadelphia, Pennsylvania
Dr. and Mrs. Sydney Jacobs, New Orleans, Louisiana
Dr. and Mrs. Minas Joannides, Chicago, Illinois
Mr. and Mrs. Murray Kornfeld, Chicago, Illinois
Dr. Milton S. Lloyd, New York, New York
Miss Harriet E. Lumm, Chicago, Illinois
Dr. and Mrs. Herbert L. Mantz, Kansas City, Missouri
Dr. Louis Mark, Columbus, Ohio
Dr. and Mrs. J. Winthrop Peabody, Washington, D. C.
Dr. and Mrs. John Roberts Phillips, Houston, Texas
Misses Dana and Ann Phillips, Houston, Texas
Dr. and Mrs. I. L. Robbins, New Orleans, Louisiana
Miss Faye Robbins, New Orleans, Louisiana
Dr. and Mrs. A. J. Steiner, St. Louis, Missouri
Dr. and Mrs. Paul A. Turner, Louisville, Kentucky
Miss Joeann Turner, Louisville, Kentucky
Dr. David Waterman, Knoxville, Tennessee

A scientific session was held at the Mercedes Hospital, Havana, on Tuesday evening. The following program was presented:

"Pneumoperitoneum," Dr. Sydney Jacobs, New Orleans, Louisiana.
"Esophageal Surgery," Dr. Minas Joannides, Chicago, Illinois.
"Histoplasmosis," Dr. Herbert L. Mantz, Kansas City, Missouri.
Motion Pictures of an Operation on the Heart, Dr. Osler Abbott, Emery, Georgia.

Dr. Antonio Rodriguez Diaz discussed Dr. Joannides' paper and presented his own cases on surgery of the esophagus. Dr. Jackson gave a brief talk on Pan American activities of the College. More than one hundred physicians attended the scientific session. Transportation to the Mercedes Hospital, a few blocks from the Hotel Nacional, was furnished by Drs. Rivero and Mendoza.

The Cuban Chapter was host at a dinner the next evening, November 6th, which was given at the Restaurant Paris in the Plaza de la Catedral. Talks were given by Dr. E. W. Hayes, Monrovia, California, Chairman of the Council on Undergraduate Medical Education of the College, Dr. J. Winthrop Peabody, Washington, D. C., Chairman of the Council on Postgraduate Medical Education, and Dr. Gustavo Aldereguia of Havana, Past President of the Cuban Chapter. Dr. Aldereguia made a brilliant

CUBAN CHAPTER MEETING, NOVEMBER 5, 1946

Scientific Session, Mercedes Hospital, Havana

Standing, left to right: Dr. Edward W. Hayes, Monrovia, California; Dr. J. Winthrop Peabody, Washington, D. C.; Dr. Chevalier L. Jackson, Philadelphia, Pennsylvania; Dr. Octavio Rivero, Havana, Cuba; Dr. Louis Mark, Columbus, Ohio; Dr. Minas Joannides, Chicago, Illinois; unidentified; Dr. Rene G. Mendoza, Havana, Cuba; Mr. Murray Kornfeld, Chicago, Illinois.

speech and concluded his talk with the following message of welcome to the delegates from the United States: "Like the old stones of the old buildings of old Havana we take you into our hearts and bid you welcome." Dr. Octavio Rivero, President of the Cuban Chapter, presided at the dinner meeting and introduced the speakers.

The Havana newspapers, El Pais and El Mundo, carried articles and photographs concerning the meeting.

Dr. Antonio Navarrete, Regent of the College for Cuba, made the arrangements for this excellent meeting with the assistance of Dr. Octavio Rivero and Dr. Rene Garcia Mendoza, President and Vice-President of the Cuban Chapter. Unfortunately, however, Dr. Navarrete was compelled to be away from Havana at the time of the meeting and his absence was deeply regretted by the College members.

Officers of the Cuban Chapter of the College are:

Dr. Octavio Rivero Partagas, President.
Dr. Rene Garcia Mendoza, Vice-President.
Dr. Orfilio Suarez de Bustamente, Secretary-Treasurer.

Past Presidents:

Dr. Alfredo Antonetti
Dr. Gustavo Aldereguia
Dr. O. Suarez de Bustamente

INDIANA CHAPTER

On October 30, 1946, a joint meeting of the Indiana Chapter of the American College of Chest Physicians and the Anti-Tuberculosis Committee of the Indiana State Medical Association was held in Indianapolis. It was a dinner meeting and was attended by approximately forty-five members and guests. The following program was carried out:

Address:

"Report of Routine Photo-Fluorographic Chest Examinations of General Hospital Admissions," Dr. Harold C. Ochsner, Indianapolis.

Discussers:

Dr. Raymond C. Beeler, Indianapolis
Dr. H. M. Draper, F.C.C.P., Fort Wayne
Dr. E. W. Custer, F.C.C.P., South Bend

During the X-Ray Conference which followed the program, there was considerable discussion about x-ray survey work.

At the business meeting the following officers were elected for the Indiana Chapter of the College:

Dr. J. V. Pace, New Albany, President
Dr. O. T. Kidder, Fort Wayne, Vice-President
Dr. H. B. Pirkle, Rockville, Secretary-Treasurer

MISSOURI CHAPTER

The Missouri Chapter of the College met in conjunction with the Southwest Clinical Conference on October 6 at Kansas City. Twenty-five members and guests attended the dinner and meeting. Dr. Samuel H. Snider, President of the Chapter called the meeting to order. The chairmen of the various committees were named.

Dr. Carl Pfuetze, F.C.C.P., Cannon Falls, Minnesota, gave an excellent and completely illustrated lecture on streptomycin with case reports.

The meeting was closed with an informal X-Ray Conference which was enjoyed by all.

NEW JERSEY CHAPTER

The Winter Scientific Meeting of the New Jersey Chapter was held on December 11 at the Jersey City Medical Center. The following program was presented:

"A Case of Cystic Disease of the Lung," Dr. Thomas DeCecio and Dr. I. E. Gerber.

"The Diagnosis of Mediastinal Tumors," Dr. Herbert C. Maier.

"A Case of Systemic Blastomycosis," Dr. Samuel Cohen.

"Spontaneous Hemopneumothorax. Complication Requiring Surgical Management," Dr. Benjamin J. Elwood.

"Penicillin in the Therapy of Pulmonary Actinomycosis," Dr. Abraham E. Jaffin.

"Case Report: Disseminated Coccidioidomycosis," Dr. Samuel Koopstein.

OHIO CHAPTER

Dr. E. E. Kirkwood, Youngstown, President of the Ohio Chapter of the College, has announced the following committee appointments:

Program Committee:

Dr. David W. Heusinkveld, Cincinnati, Chairman
Dr. Herman J. Nimitz, Cincinnati.
Dr. Louis Mark, Columbus.

Medical Education Committee:

Dr. George M. Curtis, Columbus, Chairman.
Dr. R. C. McKay, Cleveland.
Dr. John H. Skavlem, Cincinnati.

Membership Committee:

Dr. Karl P. Klassen, Columbus, Chairman.
Dr. Edgar P. Adams, Warren.
Dr. Harold G. Curtis, Cleveland.

PACIFIC NORTHWEST DISTRICT CHAPTER

Dr. Frank I. Terrill, Deer Lodge, Montana, President of the Pacific Northwest District Chapter of the College, has made the following committee appointments for the chapter:

Medical Education Committee:

Dr. Frederick Slyfield, Seattle, Washington.
Dr. William S. Conklin, Portland, Oregon.
Dr. Grover C. Bellinger, Salem, Oregon.
Dr. Irvin R. Fox, Eugene, Oregon.

Membership Committee:

Dr. John E. Nelson, Seattle, Washington.
Dr. Frank S. Miller, Spokane, Washington.
Dr. Howard L. Hull, Yakima, Washington.
Dr. J. Karl Poppe, Portland, Oregon.
Dr. W. Elliott Harrison, Vancouver, B. C.

Program Committee:

Dr. Byron F. Francis, Seattle, Washington.
Dr. Cedric Northrop, Seattle, Washington.
Dr. Orval F. Swindell, Boise, Idaho.
Dr. John S. Srail, Elma, Washington.
Dr. John F. Steele, Tacoma, Washington.

Nominating Committee:

Dr. James M. Odell, The Dalles, Oregon.
Dr. Frederick Slyfield, Seattle, Washington.
Dr. William S. Conklin, Portland, Oregon.

PERUVIAN CHAPTER

The Peruvian Chapter of the College held their Second Annual Meeting at the Dispensario Central, Lima, Peru, on November 11, 12 and 13, 1946. Dr. Ovidio Garcia Rosell, Governor of the College for Peru, and Past-President of the Peruvian Chapter, announced that the meeting was very successful. The following excellent program was presented:

November 11th:

"Imagenes redondeadas en la tuberculosis pulmonar,"
Dr. Max Espinoza Galarza, F.C.C.P., Lima.
"Algunos aspectos de la asistencia antituberculosa en el Callao,"
Dr. Luis E. Hubner, F.C.C.P., Callao.
"Asistencia dispensarial de los adolescentes,"
Dr. Victor Narvaez Obeso, Lima.
"Catastro Roentgenfotografico en el Departamento Municipal de Sanidad," Dr. Humberto Valderrama, Lima.

November 12th:

"Imagenes planigraficas de perfil,"
Prof. Dr. Ovidio Garcia Rosell, F.C.C.P., Lima.
"Cavernas no visibles a la radiografia plana,"
Dr. Leopoldo Molinari Balbuena, F.C.C.P., Lima.
"Enfermedad de Hodgkin de forma mediastinal,"
Dr. Horacio Cachay Diaz, Lima.
"Cavernas empastadas,"
Dr. Ramon Vargas Machuca, F.C.C.P., Lima.

November 13th:

"Bronconeumonias atipicas,"
Dr. Juan Escudero Villar, F.C.C.P., Lima.
"Tuberculosis en internados escolares,"
Dr. Roman del Castillo, Lima.
"Un caso de paralisis transitoria del frenico,"
Dr. Jorge Sarmiento E., Lima.

The business meeting of the Peruvian Chapter was held on the last day of the meeting and the present officers were re-elected for the coming year. The officers of the chapter are:

Dr. Max Espinoza Galarza, Lima, President.
Dr. Luis Cano Gironda, Lima, Vice-President.
Dr. Ramon Vargas Machuca, Lima, Secretary.
Dr. Luis E. Hubner, Callao, Treasurer.
Dr. Mario Pastor, Lima, Bibliothecary.

WISCONSIN CHAPTER

Dr. Alfred A. Busse, Jefferson, Wisconsin, President of the Wisconsin Chapter of the College has announced the following committee appointments:

Program Committee:
Dr. A. L. Banyai, Milwaukee, Chairman
Dr. Phillip F. Feingold, Milwaukee
Dr. Ethan Pfefferkorn, Oshkosh
Dr. John K. Shumate, Madison

General Arrangements Committee:
Dr. Mischa Lustok, Milwaukee, Chairman
Dr. Earl E. Carpenter, Superior
Dr. A. S. Kimball Jr., Madison
Dr. Leonard W. Moody, Bayfield
Dr. Valentine O'Malley, Wauwatosa
Dr. Marres H. Wirig, Madison

Educational Committee:
Dr. Ethan Pfefferkorn, Oshkosh, Chairman
Dr. H. H. Christensen, Wausau
Dr. William T. Clark, Janesville
Dr. John C. Dundee, Waukesha
Dr. Karl Kassowitz, Milwaukee

Publicity and Reception Committee:
Dr. Laurie L. Allen, Milwaukee, Chairman
Dr. G. D. Guilbert, Wood
Dr. Douglas Gutheil, Milwaukee
Dr. Paul Jahn, Milwaukee

Membership Committee:
Dr. Leon H. Hirsh, Milwaukee, Chairman
Dr. Isabel Gadzikowski, Milwaukee
Dr. William B. Ford, Milwaukee
Dr. Thomas C. Nuzum, Janesville

Scientific Arrangements Committee:
Dr. George H. Jurgens, Milwaukee, Chairman
Dr. Paul Eisele, Waukesha
Dr. John P. Fetherston, Milwaukee
Dr. Stanley R. Szymanski, Wood

X-Ray Conference Committee:

Dr. William T. Clark, Janesville, Chairman
Dr. Henry A. Anderson, Stevens Point
Dr. Harry Barrell, Waukesha
Dr. John W. Connell, Fond du Lac
Dr. Esther Goldberger, Milwaukee
Dr. Paul E. Pifer, Kenosha

Nominating Committee:

Dr. Carl O. Schaefer, Racine, Chairman
Dr. Andrew L. Banyai, Milwaukee
Dr. Alfred A. Busse, Jefferson

College News Notes

BOARD OF EXAMINERS

Oral and written examinations for Fellowship in the College were given at Miami, Florida, on November 3, 1946, at the time of the Semi-Annual Meeting of the Board of Regents. Dr. Alvis E. Greer, Chairman of the Board of Examiners, conducted the examinations with Dr. E. W. Hayes and Dr. Minas Joannides assisting.

The following candidates for Fellowship successfully passed the oral and written examinations:

W. W. Coulter, M.D., Sanatorium, Texas.
Sheldon E. Domm, M.D., Knoxville, Tennessee.
P. M. Huggin, M.D., State Park, South Carolina.
Timothy Liang, M.D., Cassadaga, New York.
Walter Raab, M.D., Glenn Dale, Maryland.
Leon Ross, M.D., Brecksville, Ohio.
E. A. Sindel, M.D., Bethlehem, Pennsylvania.
Morris M. Snyder, M.D., Chicago, Illinois.

The questions used in the written examinations were as follows:

Thoracic Diseases (Medical Aspects)

Part I

Discuss the pathogenesis of pulmonary emphysema.

Part II

(Answer 3 of the 5 questions)

(1) Discuss the differential diagnosis of early pulmonary carcinoma and early pulmonary tuberculosis.

(2) Outline the important steps in the treatment of active clinical or secondary pulmonary tuberculosis in a young adult as they should be carried out until the patient regains his health. Give the important factors which might indicate the various steps which may be used in the treatment of such a case.

(3) Discuss the contraindications for artificial pneumothorax.

(4) Consulted by a female patient 42 years old, married and with no children, who gives a history of always having been well previously and apparently happy, who complains of the following symptoms: sleeps poorly for the past three or four years; tired with lack of endurance; some coughing and hacking; raises a dram or so of sputum in 24 hours, mostly mucoid; has lost 4 pounds the past month; menses regular and

normal. Patient states that she smokes a package of cigaretts a day, takes a highball occasionally. The onset of symptoms, except insomnia, was 5 months previous to the date of examination. X-ray pictures of the chest (lungs) are negative. Sedimentation test is 7 mm. in one hour. There are no other complaints or abnormal physical findings. What would be your advice to this patient?

(5) Discuss briefly the difference between primary and secondary tuberculosis from a pathological, diagnostic and therapeutic standpoint.

Thoracic Diseases (Surgical Aspects)

(Answer No. 1, and any 2 of the Following questions)

(1) How do you manage a case of tuberculous empyema complicated by bronchopleural fistula?

(2) Discuss primary carcinoma of the lung from:
- (a) the standpoint of suggestive or presumptive signs;
- (b) positive evidence;
- (c) how would you obtain positive evidence?

(3) Discuss spontaneous pneumothorax including the diagnosis and management.

(4) Discuss the relative merit of the various permanent forms of collapse therapy:
- (a) what are your indications for thorocoplasty?
- (b) what are your contraindications for a thorocoplasty?

(5) Discuss the indications for resection in pulmonary tuberculosis.

Bacteriology and Immunology

(Answer question 1 and 2, and one of the following three questions)

(1) Discuss briefly the characteristics of the tubercle bacillus. Give the different types and their relation to human tuberculosis. What are the ordinary means by which the different types of this bacillus are distinguished?

(2) List fungus disease that may involve the lung and briefly describe fungi involved.

(3) Give currently employed typing procedure for pneumococci. Which types are most commonly recovered from cases of lobar pneumonia?

(4) List in order of their frequency the organisms found responsible for cases of bronchopneumonia.

(5) List the more common non-tuberculous chronic diseases of the lungs (five or six) in which the presence or absence of specific organisms is important in differential diagnosis. Briefly outline differential diagnosis of each of these from pulmonary tuberculosis.

Pathology

(Answer any three of the following five questions)

(1) Name the pneumoconioses and describe the findings in the three most important dust diseases.

(2) Describe briefly the chain of pathological events involved in the spread of tubercle bacilli from an active focus in the lung to other parts of the same lung; to the other lung or to other parts of the body.

(3) Give the pathogenesis of lung abscess.

(4) List the tumors which may arise in the mediastinum.

(5) Describe briefly the different pathological processes that may take place as active tuberculous foci in the lungs retrogress and eventually become inactive.

Physiology

(Answer any two of the following five questions)

(1) Define and discuss vital capacity. How is vital capacity determined, and why is it important in the treatment of tuberculosis?

(2) How would you advise as to possible bad effects that may be encountered in each of the following patients who is contemplating an airplane trip during which travel may be at an altitude of 10,000 feet?
- (a) An active case of pulmonary tuberculosis.
- (b) An arrested case of pulmonary tuberculosis, particularly if there is fairly extensive fibrosis.

(c) A patient with unilateral artificial pneumothorax.
(d) A patient who has had a seven rib extrapleural thorocoplasty.
Give briefly the reasons for your advice in each case.

(3) Would oxygen therapy be indicated or not in the presence of chronic cyanosis? Explain.

(4) What are the current views relative to the mechanism of precipitation of atelectasis?

(5) What are the possible physiological explanations of the characteristic fever associated with multiple small pulmonary emboli?

Anatomy of the Chest

(Answer any two of the following three questions)

(1) Discuss the blood supply of the lungs and state practical applications attendant thereto.

(2) Discuss surface anatomy of the various lobes and fissures of the lungs.

(3) Discuss the lymphatic system of the thoracic cage.

COUNCILS AND COMMITTEES

New members have been appointed to many of the College councils and committees to replace those whose terms of service have expired. We are pleased to list below the present membership of some of the councils and committees:

Council on Undergraduate Medical Education:

E. W. Hayes, M.D., Monrovia, California, Chairman
Andrew L. Banyai, M.D., Milwaukee, Wisconsin
Benjamin L. Brock, M.D., Oteen, North Carolina
Louis L. Friedman, M.D., Birmingham, Alabama
Sydney Jacobs, M.D., New Orleans, Louisiana
C. Howard Marcy, M.D., Pittsburgh, Pennsylvania
Elliott Mendenhall, M.D., Dallas, Texas
Nelson W. Strohm, M.D., Buffalo, New York

Council on Postgraduate Medical Education:

J. Winthrop Peabody, M.D., Washington, D. C., Chairman
Seymour M. Farber, M.D., San Francisco, California
Frank R. Ferlaino, M.D., New York, New York
Alvis E. Greer, M.D., Houston, Texas
Chevalier L. Jackson, M.D., Philadelphia, Pennsylvania
I. L. Robbins, M.D., New Orleans, Louisiana
Moses J. Stone, M.D., Boston, Massachusetts
Willard Van Hazel, M.D., Chicago, Illinois
W. Bernard Yegge, M.D., Denver, Colorado

Council of Medical Directors and Superintendents of Tuberculosis Hospitals and Sanatoria:

Benjamin L. Brock, M.D., Oteen, North Carolina, Chairman
Russell S. Anderson, M.D., Erie, Pennsylvania
John B. Andosca, M.D., Mattapan, Massachusetts
Merle D. Bonner, M.D., Jamestown, North Carolina
E. F. Conlogue, M.D., Dayton, Ohio
M. A. Cunningham, M.D., Beaumont, Texas
Stephen A. Douglass, M.D., Paterson, New Jersey
David F. Loewen, M.D., Decatur, Illinois
Harry C. Warren, M.D., San Francisco, California

Sub-Committee on Sanatorium Standards:

I. D. Bobrowitz, M.D., Otisville, New York, Chairman
Russell S. Anderson, M.D., Erie, Pennsylvania
Philip H. Narodick, M.D., Seattle, Washington
Arnold Shamaskin, M.D., Hines, Illinois
Charles F. Taylor, M.D., Norton, Kansas
B. H. Wardrip, M.D., San Jose, California

Sub-Committee on Rehabilitation:

Allan Hurst, M.D., Denver, Colorado, Chairman
E. F. Conlogue, M.D., Dayton, Ohio
Myron Herman, M.D., New York, New York
Herman E. Hilleboe, M.D., Washington, D. C.
C. Gerald Scarborough, M.D., San Jose, California
Arthur S. Webb, M.D., Glen Ellyn, Illinois

National Council of Tuberculosis Committees:

James H. Stygall, M.D., Indianapolis, Indiana, Chairman
James F. Brewer, M.D., New Bedford, Massachusetts
Maurice Campagna, M.D., New Orleans, Louisiana
Cole B. Gibson, M.D., Meriden, Connecticut
D. W. Heusinkveld, M.D., Cincinnati, Ohio
John S. Packard, M.D., Allenwood, Pennsylvania
Rufus A. Schneiders, M.D., San Diego, California
Nelson W. Strohm, M.D., Buffalo, New York
Darrell H. Trumpe, Springfield, Illinois

Council on Pan Pacific Affairs:

Harry C. Warren, M.D., San Francisco, California, Chairman
Capt. Robert E. Duncan, M.C., U.S.N., Hawaiian Islands, Vice-Chmn.
Indubhusan Basu, M.D., Calcutta, India
John S. Bouslog, M.D., Denver, Colorado
Miguel Canizares, M.D., Quezon City, Philippine Islands
A. Barklie Coulter, M.D., Washington, D. C.
John Bell Ferguson, M.D., Melbourne, Australia
W. Elliott Harrison, M.D., Vancouver, British Columbia
Paul H. Holinger, M.D., Chicago, Illinois
A. Holmes Johnson, M.D., Kodiak, Alaska
Maj. Gen. S. U. Marietta, Washington, D. C.
J. Ancheng Miao, M.D., Kunming, China
George G. Ornstein, M.D., New York, New York
Richard H. Overholt, M.D., Brookline, Massachusetts
Lincoln Pan, M.D., Shaoshing, China
Li Shu Fan, M.D., Hong Kong, China

Board of Examiners:

Alvis E. Greer, M.D., Houston, Texas, Chairman
Edward W. Hayes, Monrovia, California
Alton Ochsner, M.D., New Orleans, Louisiana

Council on Public Health:

Paul A. Turner, M.D., Louisville, Kentucky, Chairman
Sidney A. Britten, Comdr., M.C., U.S.N., Washington, D. C.
Richard Davison, M.D., Chicago, Illinois

John B. Grow, Col., M.C., Denver, Colorado
W. H. Hatfield, M.D., Vancouver, British Columbia
Herman E. Hilleboe, M.D., Washington, D. C.
Samuel E. Thompson, M.D., Kerrville, Texas
Walter E. Vest, M.D., Huntington, West Virginia
Roy A. Wolford, M.D., Washington, D. C.

Council on Public Relations:

John Roberts Phillips, M.D., Houston, Texas, Chairman
Grant Thorburn, M.D., New York, N. Y., Vice-Chairman
R. B. Homan, M.D., El Paso, Texas
Everett Morris, M.D., Olive View, California
J. Karl Poppe, M.D., Portland, Oregon

The Milwaukee Metropolitan Section of the American College of Chest Physicians held an informal dinner meeting at the Medford Hotel on Friday, November 29th. Dr. Stanley Szymanski presented the subject "Pneumoperitoneum". There was a large attendance and a stimulating discussion.

Dr. William A. Hudson, F.C.C.P., Detroit, Michigan, was recently appointed by Dr. Charles M. Hendricks, President, to serve as Historian of the American College of Chest Physicians.

Dr. Enrique Coronado Iturbide, F.C.C.P., Guatemala City, has been appointed to serve as Governor of the College for Guatemala.

Dr. Carlos Gonzales B., F.C.C.P., San Salvador, has accepted appointment to serve as Governor of the College for El Salvador.

DR. HERMAN E. HILLEBOE APPOINTED TO SUCCEED DR. LEWIS R. THOMPSON

After 36 years of distinguished service in the United States Public Health Service, Dr. Lewis Ryers Thompson, Assistant Surgeon General in the Bureau of State Services was retired for medical disability, on November 1st. Dr. Thompson's resignation brings to a close a brilliant record in the history of the United States Public Health Service. Dr. Thompson joined the Public Health Service as an Assistant Surgeon in 1910. He served in all the ranks of the service and was appointed Assistant Surgeon General in 1930, a position which he held for 16 years. Since 1943 he has served as Chief and Associate Chief of the Bureau of State Services in which capacity he supervised the activities of the States Relations Division, Venereal Disease Division, Industrial Hygiene Division, Tuberculosis Control Division, and Public Health Nursing.

To fill the vacancy left by Dr. Thompson, Dr. Herman E. Hilleboe, who has been Chief of the Tuberculosis Control Division since its in-

ception in 1944, has been appointed Associate Chief of the Bureau of State Services, with the rank of Assistant Surgeon General. Dr. Hilleboe has been engaged in public health work since 1933 and has specialized in tuberculosis control. He was assigned by the Public Health Service to study tuberculosis control in England, the Scandinavian countries, Germany, and France. He has served in many important capacities on special committees on tuberculosis here and abroad. In his new position Dr. Hilleboe will have supervisory responsibility for the Tuberculosis Control Division and the newly created Hospital Facilities Division. In the latter capacity he will assist in the administration of the recently enacted Hospital Construction Act.

Senior Surgeon Francis J. Weber, formerly Assistant Chief of the Tuberculosis Control Division, has been promoted to the rank of Medical Director and will serve as Chief of the Tuberculosis Control Division. Dr. Weber has had extensive experience in public health work in Georgia, Louisiana, Michigan, and California. He assisted in the establishment of the Tuberculosis Control Division and has served as Medical Officer in Charge of the State Aid Section of the Division since 1944. In this position Dr. Weber administered grant-in-aid funds distributed to the States by the Tuberculosis Control Division.

VETERANS ADMINISTRATION
BRANCH SECTION CHIEFS IN THORACIC SURGERY

	Branch
Dr. Gustaf Lindskog Yale University Medical School, New Haven, Connecticut.	No. 1
Dr. Warriner Woodruff 8 Church Street, Saranac Lake, New York.	No. 2
Dr. Edward M. Kent University Club, Pittsburgh, Pennsylvania.	No. 3
Dr. Everett Drash University of Virginia Hospital, Charlottesville, Virginia.	No. 4
Dr. Edward F. Parker Jr. 70 Hasell Street, Charleston, South Carolina.	No. 5
Dr. William M. Tuttle 307 David Whitney Building, Detroit, Michigan.	No. 6
Dr. Joseph Gale University of Wisconsin, Madison, Wisconsin.	No. 7
Dr. O. T. Clagett Mayo Clinic, Rochester, Minnesota.	No. 8
Dr. Thomas Burford Department of Surgery, Barnes Hospital, St. Louis 10, Missouri.	No. 9
Dr. Michael DeBakey Tulane Medical School, New Orleans, Louisiana.	No. 10
Dr. Oscar C. Procter Medical Dental Building, Seattle, Washington.	No. 11
Dr. Paul Samson 2938 McClure, Oakland, California.	No. 12
Colonel John B. Grow Fitzsimons General Hospital, Denver, Colorado.	No. 13

ANNOUNCEMENT

The Scientific Program Committee, American College of Chest Physicians, will be pleased to receive manuscripts or abstracts for approval by the committee in connection with the 13th Annual Meeting of the College to be held in Atlantic City in June 1947. Please address all communications to the Chairman of the Committee.

Andrew L. Banyai, M.D., Chairman, Scientific Program Committee
Muirdale Sanatorium, Milwaukee 13, Wisconsin

Burgess Gordon, M.D.
Philadelphia, Pennsylvania

Minas Joannides, M.D.
Chicago, Illinois

ANNOUNCEMENT

Reprints of the "General Code Relating to the Control of Tuberculosis by Modern Means" are available for distribution upon request at the Executive Offices of the American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois. This outline was prepared by the Committee on State Laws of the College and published in the September-October issue of "Diseases of the Chest".

MEMBERS OF THE COLLEGE RELEASED FROM THE ARMED SERVICES

Albertson, L. C., Lt. Comdr., MC, Veterans Hospital, Outwood, Kentucky.
Alley, Ralph Martin, Major, MC, Indiana Sanatorium, 801 North Vassar, Albuquerque, New Mexico.
Amazon, Peter, Capt., MC, 1040 Sterling Place, Brooklyn, New York.
Bachman, Henry, 1st Lt., MC, 157 North Union, Delaware, Ohio.
Baker, Robert Henry, Lt. Comdr., MC, Belmont Hospital, Worcester, Mass.
Banquer, Jacob Ellis, Colonel, MC, 598 Washington St., Brookline, Mass.
Bartholomew, Jack D., Major, MC, 701 Seventh Street, Boulder, Colorado.
Bass, Hyman E., Bapt., MC, 266 West End Avenue, New York, New York.
Beloff, Harry, Capt., MC, 844 Ritner St., Philadelphia, Pennsylvania.
Beloff, Lewis, Capt., MC, 844 Ritner St., Philadelphia, Pennsylvania.
Blades, Brian B., Lt. Col., MC, 1335 "H" Street, Washington, D. C.
Bloomberg, Allan E., Capt., MC, 214 W. 72nd St., New York 23, New York.
Blum, Milton, Capt., MC, 310 Stigman Parkway, Jersey City, New Jersey.
Bornstein, Paul K., Major, MC, 320 Asbury Ave., Asbury Park, New Jersey.
Bridge, Frederick D., Capt., MC, 205 Avenue "P", Brooklyn, New York.
Brotman, David M., Lt., MC, USNR, 5632 Sheridan Road, Chicago, Illinois.
Bruhl, Charles K., Capt., MC, 1706 North Blvd., Houston, Texas.
Bugden, Walter F., Capt., MC, 677 W. Onondago St., Syracuse, New York.
Castle, Charles A., Lt. Comdr., MC, Dunham Hospital, Cincinnati 15, Ohio.
Chandler, John H., Major, MC, 706 S. Mansfield, Memphis, Tennessee.
Cabb, Norman E., Lt. Comdr., MC, Colais, Maine.
Cohen, Samuel C., Lt., MC, 416 Marloborugh St., Boston, Massachusetts.
Combs, Stuart Richardson, Capt., MC, 2620 N. 10th St., Terre Haute, Ind.
Dailey, J. Emerson, Lt. Comdr., MC, 3614 Tangley St., Houston 5, Texas.
Davis, John Dwight, Lt. Col., MC, 1930 Wilshire Blvd., Los Angeles 5, Cal.
Davis, Paul Vincent, Major, MC, 94 Main Street, Bridgton, Maine.

Deason, Lloyd D., Lt. Comdr., MC, 301 Rogers St., Henderson, Texas.
Dimsdale, Lewis J., Capt., MC, 431-34 Frances Bldg., Sioux City, Iowa.
Domm, Sheldon E., Lt. USNR, MC, 529 E. Chicago Ave., Neperville, Illinois.
Drozda, Joseph P., Lt. Col., MC, 510 Redick Tower Bldg., Omaha, Neb.
Dundee, John C., Capt., MC, Veterans Hospital, Waukisha, Wisconsin.
Eckstein, Albert, Major, MC, 1514 N. 10th Street, Phoenix, Arizona.
Evans, George Francis, Major, MC, 206 N. Chestnut St., Clarksburg, W. Vir.
Feinberg, Abraham, Lt. Comdr. USNR, MC, 5860 Kenmore Chicago, Ill.
Felts, Clifton, Capt. MC, 1011 Roosevelt, Seminole, Oklahoma.
Felson, Henry, Capt., MC, 732 Greenwood Avenue, Cincinnati, Ohio.
Fisher, Martin M., 1st Lt., MC, 45 Linden Blvd., Brooklyn, New York.
Flattery, James F., Capt., MC, 575 W. 172nd St., New York 32, New York.
Frost, Russell Harold, Comdr., MC, Veterans Administration, Midland Bank Building, Minneapolis, Minnesota.
Galinsky, Leon J., Capt., MC, Broadlawns Polk Company Hospital, 18th and Hickman Road, Des Moines, Iowa.
Gibbons, Morton R. Jr., Comdr. USNR, MC, 3979 Washington Street, San Francisco 18, California.
Gloeckler, Bernhard B., Capt., MC, Mt. Pleasant, Iowa.
Goldman, Alfred, Lt. Comdr., MC, 516 Sutter St., San Francisco, Calif.
Goliger, J. J., Lt., MC, 3506 Newkirk Avenue, Brooklyn, New York.
Gompertz, John L., Major, MC, No. 1 Hacienda Road, Orinda, California.
Gorfinkel, Leon H., Comdr., MC, 18225 Beverly Glen Boulevard, Los Angeles 26, California.
Gorlin, David O., Major, MC, 130-01 Leffests Boulevard, South Ozone Park, Queens, New York.
Gorlin, Philip Samuel, Capt., MC, 130-01 Lefferts Boulevard, South Ozone Park, Queens, New York.
Gould, Arthur A., Major, MC, 1215 Ocean Avenue, Santa Monica, Calif.
Greco, Edward A., Major, MC, 12 Pine Street, Portland, Maine.
Grund, Joseph, Capt., MC, 7 Low Street, Liberty, New York.
Guzman, Manuel Jr., Major, MC, Santurce, Porto Rico.
Hall, Snowden C. Jr., Lt. Comdr., MC, Hawthorne Drive, Forest Hills, Daneville, Virginia.
Hanna, Roger J., Lt. Col., MC, 1301 Washington Ave., Jackson, Michigan.
Hardie, Philip W., Lt. Col., MC, 229 Herkimer St., Hamilton, Ontario.
Harris, Marvin Saul, Capt., MC, 2007 Wilshire Blvd., Los Angeles, Calif.
Harrison, David A., Capt., MC, Broadacres Sanatorium, Utica, New York.
Heinrich, J. Fuhrman, Lt. Comdr., MC, 43-55 Kissens Blvd., Flushing, N. Y.
Hellweg, Charles Edward, Major, MC, Pierce City, Missouri.
Henry, Russell Seldon, Lt. Comdr., MC, 1019 Hume Mansur Building, Indianapolis, Indiana.
Hill, Vivian D., Major, MC, 118 South Street, Mobile, Alabama.
Hudson, William E., Capt., MC, 131 Fair Ave., N. E., New Philadelphia, O.
Hughes, S. Edwin, Comdr. USNR, MC, U.S. Veterans Hospital, Oteen, North Carolina.
Idstrom, Linneus G., Lt. Col., MC, Care Dr. I. G. Idstrom, Minnesota State Sanatorium, Walker, Minnesota.
Ingegno, Alfred P., Major, MC, 880 Carrol Street, Brooklyn, New York.
Jones, John Ben, Lt. USNR, MC, 115 N. Gex St., La Plata, Missouri.
Kellogg, Howard B., Lt. Col., MC, 2001 24th Ave., N. Seattle, Washington.
Kent, Edward M., Comdr., MC, Allegheny Gen. Hospital, Pittsburgh, Pa.
Kincov, Jacob, Major, MC, 243 Spring Garden St., Easton, Pennsylvania.

King, Richard, Capt., MC, 261 Alberta Drive, N. E., Atlanta, Georgia.
Kirshner, Jacob Jesse, Capt., MC, 2025 Spruce St., Philadelphia 3, Pa.
Klein, William S., Capt., MC, 4424-A Drexel Blvd., Chicago, Illinois.
Klupt, Robert Louis, Major, MC, 5 East 76th St., New York 21, New York.
Knies, Phillip T., Lt. Col., MC, 463 E. Town Etreet, Columbus 4, Ohio.
Knoepp, Louis F., Lt. Col., MC, 5900 Line Ave., Shreveport, Louisiana.
Konterwitz, Harry, Major, MC, 41-22 42nd St., Sunnyside, Long Island.
Lande, Frank, Major, MC, Rocky Glen Sanatorium, McConnelsville, Ohio.
Larson, Swen Leonard, Major, MC, 213 Columbia St., Elmira, New York.
Lederer, Francis L., Lt. Comdr., MC, 307 N. Michigan Ave., Chicago, Ill.
Lichtenberg, Walter, Capt., MC, 116 Central Park South, New York, N. Y.
Lipstein, Samuel, Capt., MC, Workmen's Circle San., Liberty, New York.
Lovgren, Robert Ellsworth, Capt., MC, McMillan Hospital, 600 S. Kingshighway, St. Louis, Missouri.
Madding, Gordon F., Major, MC, 111 E. Harris Ave., San Angelo, Texas.
Marcus, David, Capt., MC, 1285 Addison Road, Cleveland 3, Ohio.
Mayo, Leroy E., Capt., MC, Holden Clinic, Holden, Massachusetts.
McDaniel, Walter Shaw, Lt. Comdr., MC, 2231 Southgate, Houston, Texas.
Mehmert, Henry Eugene, Major, MC, Oleng, Illinois.
Mellies, Chester J., Lt. Col., MC, Care Stalleup Bldg., Sikiston, Missouri.
Milanesi, Armand M., Major, MC, 2 Potter Place, Weehawken, New Jersey.
Miller, Morell Waldo, Lt. Comdr., MC, 332 Audobon Bldg., New Orleans, La.
Miller, Raymond Everett, Lt., MC, 192 W. State Street, Trenton, N. J.
Minnis, Dean Hugh, Capt., MC, Pleasant View Sanitarium, Amherst, Ohio.
Morris, Stanley F., Lt., MC, 246 Roswell St., Long Beach 3, California.
Mosier, Dwight J., Capt., MC, 307 W. Midland St., Bay City, Michigan.
Myers, Dan Wilbur, Lt. Col., MC, 323 Michigan Central Terminal, Detroit 16, Michigan.
Narodick, Philip Howard, Major, MC, 2229 22nd Ave. North, Seattle, Wash.
Nathan, David A., Major, MC, 605 Lincoln Road, Miami Beach, Florida.
Nimitz, Herman J., Lt. Col., MC, Hamilton County Tuberculosis Hospital, Cincinnati, Ohio.
O'Hara, Francis Paul, Comdr. USNR, MC, 233 "A" St., San Diego, Calif.
O'Malley, James E., Capt., MC, Anchorage, Alaska.
Passalacqua, Luis A., Lt. Col., MC, 15 Travieso St., Santurce, Puerto Rico.
Pau, Jaime F., Capt., MC, Box 18, Hato Rey, Puerto Rico.
Paul, S. Barre, Lt. Comdr., MC, 450 Sutter St., San Francisco, California.
Piatt, Arnold D., Capt., MC, 44 East Church Street, Columbus, Ohio.
Placak, Joseph Charles Jr., Comdr., MC, 10515 Carnegie Ave., Cleveland 6, Ohio.
Plumer, Joseph Neilson, Capt., MC, Chief of Medical Service, Veterans Administration Hospital, Tucson, Arizona.
Price, Henkel Moser, Capt., MC, 205 Union Street, Wytheville, Virginia.
Reiss, Jack, Capt., MC, Veterans Administration, Indianapolis, Indiana.
Reyes, Felix M., Capt., MC, Bayamon District Hospital, Bayamon, P. R.
Robbins, Eric Patterson, Major, MC, Brookhaven, Massachusetts.
Rogers, Galen Alonzo, Major, MC, 718 Sycamore Street, Clarkston, Wash.
Rogoff, Jacob, Lt. Comdr., MC, 25 Eastern Parkway, Brooklyn, New York.
Rosenbaum, Joseph George, Capt., MC, North Royalton, Ohio (P. O. Brecksville).
Rothermich, Norman O., Capt., MC, 370 East Town St., Columbus, Ohio.
Rouff, Elliot Arnold, Lt. Comdr., MC, 1930 Wilshire Blvd., Los Angeles, Cal.
Rutherford, Findlay D., Major, MC, 1315 Emory St., San Jose 11, Cal.

Ryan, Raymond Charles, Capt., MC, 87-26 175th St., Jamaica 3, New York.
Samson, Paul Curkeet, Major, MC, 805 Highland Ave., Piedmont, Cal.
Schall, James A., Capt., MC, 5125 Leona Drive, Cincinnati, Ohio.
Schell, Robert E., Capt., MC, Bowling Green, Kentucky.
Schneble, Richard C., Lt. Comdr., MC, 401 Greenmount Blvd, Dayton 9, O.
Schock, H. Charles, Major, MC, 10515 Carnegie Ave., Cleveland 6, Ohio.
Schwartz, Benjamin, Capt., MC, Veterans Administration Facility, 130 W. Kingsbridge, Bronx, New York.
Schwartzman, Joel J., Capt., MC, 5410 Netherland Ave., Bronx 63, N. Y.
Seelig, Charles A., Lt. Comdr., MC, 33-17 70th Street, Jackson Heights, Long Island, New York.
Selman, Morris, Capt., MC, 2605 Fulton Street, Toledo, Ohio.
Shapiro, David, Capt., MC, 30 West 29th Street, Bayonne, New Jersey.
Shipp, David Harvey, Lt. Comdr., MC, 443 Donaghey Building, Little Rock, Arkansas.
Silverglade, Alexander, Capt., MC, 403 South Orange Grove Avenue, Los Angeles 26, California.
Simpson, Neill, Major, MC, 1724 Austin Avenue, Waco, Texas.
Sims, John Arthur, Lt. Col., MC, 480 Lowell Ave., Newtonville, Mass.
Sokol, Louis Irwin, Major, MC, 984 Third Ave., Los Angeles 6, California.
Spalding, William C., Lt. Col., MC, 947 W. 8th St., Los Angeles, California.
Stacey, John Wallace, Capt., MC, 1930 Wilshire Blvd., Los Angeles, Cal.
Staff, Robert A., Major, MC, Indiana State Sanatorium, Rockville, Ind.
Steen, William B., Major, MC, 110 South Scott St., Tucson, Arizona.
Steinberg, Israel, Lt. Comdr., MC, 15 W. 81st St., New York, New York.
Stork, Walter J., Capt., MC, 3801 Fannin Street, Houston, Texas.
Stickler, Cyrus W. Jr., Major, MC, 21 Brookhaven Drive, N. E., Atlanta, Ga.
Swasey, Lloyd K., Lt. Comdr., MC, 15 E. Monroe St., Phoenix, Arizona.
Swett, Herbert C., Capt., MC, City Hospital, 1515 Lafayette Avenue, St. Louis, Missouri.
Taymor, Joseph, Lt. Comdr., MC, 6777 Clyde Ave., Chicago 49, Illinois.
Tebrock, Harry E., Lt. USNR, MC, 730 Decatur Street, Brooklyn, New York.
Tennant, Raymond Elwodd, Lt. Col., MC, 1103 Medical Dental Building, Seattle 1, Washington.
Theodos, Peter Andrew, Capt., MC, 3901 "J" St., Philadelphia, Pa.
Thomas, Efton J., Capt., MC, 835 Lincoln Road, Miami Beach, Florida.
Toomey, L. O., Comdr., MC, 544 E. Main St., Bowling Green, Kentucky.
Tsoulos, George D., Capt., MC, 10 N. Clark St., Chicago, Illinois.
Van Der Schouw, Harold Mundell, Capt., MC, Lutheran Sanatorium, Wheat Ridge, Colorado.
Walters, Henry W., Colonel, MC, Sunmount, New York.
Ward, James A., Comdr., MC, 210 E. 8th St., Metropolis, Illinois.
Weinberger, Emanuel M., Major, MC, 255 S. 17th St., Philadelphia, Pa.
Weiner, Aaron A., 1st Lt., MC, 2084 61st St., Brooklyn, New York.
Weissman, Herman, Capt., MC, Veterans Administration Facility, Legion, Texas.
Wheeler, Daniel Wilbur, Capt., MC, 717 Medical Arts Building, 324 West Superior Street, Duluth, Minnesota.
Wilen, Carl John, Major, MC, Nelson Clinic, Manhattan, Kansas.
Witt, Joseph J., Lt. Comdr., MC, 258 Genesee St., Utica, New York.
Wright, Fletcher J., Major, MC, 1617 Blair Road, Petersburg, Virginia.
Yntema, Stuart, Lt. Col., MC, 333 S. Jefferson Ave., Saginaw, Michigan.
Zelman, Julius, Major, MC, 1533 Mt. View, San Bernardino, California.

Obituaries

JOHN S. AGAR JR.

1909 - 1946

Dr. John S. Agar Jr. of Little Rock, Arkansas, died suddenly of coronary occlusion on October 15, 1946.

Dr. Agar received his pre-medical education at Washington University, St. Louis, Missouri. He was graduated from the Arkansas School of Medicine. His rotating internship was served at the Jewish Hospital, St. Louis, Missouri. He served his Assistant Residency in Otology, Laryngology and Rhinology at Barnes Hospital, St. Louis, Missouri. During World War II he served in the Navy. He was attached to the 2nd Marine Division and received a personal citation for bravery.

He was a member of the American College of Chest Physicians, the Arkansas Medical Society, and Pulaski County Medical Society.

J. D. Riley, M.D.
Governor for Arkansas.

CLYDE M. FISH

1875 - 1946

With deep sorrow but humbly submitting to the will of God we report the death of Dr. Clyde M. Fish which occurred on November 21st as a result of cerebral accident.

Dr. Fish was a valued member of the New Jersey Chapter of the College, of which he was President in 1944. He was born in Bath, Pennsylvania in 1875 and was educated at Lehigh University, Rush Medical College and the Jefferson Medical College from which he graduated in 1900. In 1901 he was appointed to the staff of the Atlantic City, New Jersey Hospital and in 1916 was made Medical Director of the Atlantic City Hospital for Chest Diseases which position he held until his death. He was for many years a Director of the New Jersey Tuberculosis League.

Martin H. Collier, M.D.
Governor for New Jersey.

ANNOUNCEMENT

May 1st 1947 is the deadline for entering the $34,000 prize art contest on the special subject of "Courage and Devotion Beyond the Call of Duty" (on the part of physicians in war and in peace). This contest is open to all M. D.'s in the Western Hemisphere. The exhibition will take place in conjunction with the A. M. A. Centennial Session at Atlantic City, June 9-13th, 1947. For complete information, write or wire now to Francis H. Redewill, M.D., Secretary, American Physicians Art Association, Flood Building, San Francisco, California, or to the sponsor, Mead Johnson & Company, Evansville 21, Indiana, U.S.A.

Positions Wanted and Available

MEDICAL SERVICE BUREAU

In accordance with a resolution adopted by the Board of Regents of the College at their annual meeting held in Chicago on June 17, 1945, a Medical Service Bureau has been established at the Executive Offices of the College for the purpose of serving the members of the College being released from the armed forces.

The Bureau would appreciate receiving information from the medical superintendents of sanatoria regarding positions available at their institutions, together with full particulars as to the type of position and salary offered. Fellows of the College who are looking for assistants should send complete information to the Bureau.

Physicians being released from the armed forces who are seeking appointments and positions should send complete information to the Bureau regarding their training and the type of position desired.

Please direct all correspondence to the Medical Service Bureau, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

POSITIONS AVAILABLE

Assistant physician wanted in tuberculosis hospital in southeast Texas. Three thousand a year with complete maintenance to single man. Excellent opportunity to learn chest surgery and other collapse therapy. Good climate the year around. For further information please write Box 148A, American College of Chest Physicians, 500 N. Dearborn Street, Chicago 10, Illinois.

Staff physician wanted at approved hospital, complete medical and surgical service, out-patient facilities, tuberculosis and other chest diseases. For further information please write Box 149A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Single doctor wanted as resident physician in tuberculosis sanatorium. For further information please write Box 151A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Illinois.

Resident physician wanted at approved tuberculosis sanatorium for the training of residents in tuberculosis. Salary offered will depend on the experience and qualifications of applicant. Applicants should give their training, experience, age, etc. in their first letter and, if possible, enclose a photograph. Please address Box 152A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.

Physician wanted for medical staff of the State Tuberculosis Sanatorium, Marianna, Florida, opened September 2, 1946, with 200 bed capacity. The salary is $300.00 or more depending upon experience, with full maintenance for self and family. Please address Dr. W. D. Rosborough, Superintendent and Medical Director of the sanatorium.

POSITIONS WANTED

Fellow of the College, with 15 years full time hospital and sanatorium experience in tuberculosis and chest diseases, desires Medical Directorship in progressive institution with good clinical opportunities. For additional information please address Box 232A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.

Life Membership

AMERICAN COLLEGE OF CHEST PHYSICIANS

In view of the increase in annual dues from $10.00 per year to $15.00 per year, it is recommended that the Schedule of Life Membership Fees be revised to include this increase.

THEREFORE, BE IT RESOLVED, that the Board of Regents of the American College of Chest Physicians, hereby approve the following revised Schedule of Life Membership Fees, to become effective immediately.

SCHEDULE OF LIFE MEMBERSHIP FEES

58 years	$165.00	52 years	$255.00	45 years	$360.00
57 years	180.00	51 years	270.00	44 years	375.00
56 years	195.00	50 years	285.00	43 years	390.00
55 years	210.00	49 years	300.00	42 years	405.00
54 years	225.00	48 years	315.00	41 years	420.00
53 years	240.00	47 years	330.00	40 years	435.00
		46 years	345.00		

Minimum fee for Life Membership of Members having attained the age of 59 years or over will be $150.00.

The above figures have been estimated on a 69 year life expectancy.

APPLICATION

AMERICAN COLLEGE OF CHEST PHYSICIANS
500 North Dearborn Street
Chicago 10, Illinois

I hereby subscribe to Life Membership in the *American College of Chest Physicians*. According to the present plan, at my age the total Life Membership Fee is ($.....) .....Dollars

My annual dues shall cease at once. I shall be entitled to the publications of the College, including the Directory and the journal, *Diseases of the Chest*, without further cost, for the balance of my life, and my membership shall be permanently active.

The principal of my subscription shall be added to the permanent Endowment Fund of the College.

Name.....

Address.....

City..... Zone No..... State.....

Date of Birth..... Date of Subscription.....

Books, Reprints and Reports Received

"X-Ray Examination of the Chest in a County Hospital," Arthur Bruce Steele, M.D., Santa Barbara, California.

"Indicacao e oportunidade do Tratamento Cirurgico No Abscesso Pulmonary Agudo," Dr. Jesse Teixeira, Rio de Janeiro, Brazil.

"Twenty-Third Annual Report of the Macon County Tuberculosis Sanatorium," David F. Loewen, M.D., Decatur, Illinois.

"The Rehabilitation Program at The Municipal Sanatorium," I. D. Bobrowitz, M.D., and Joseph Newman, M.D., Otisville, New York.

"Penicillin in the Treatment of Bronchiectasis," I. D. Bobrowitz, M.D., James S. Edlin, M.D., Sydney Bassin, M.D., and J. Stanley Woolley, M.D.

"The Early Diagnosis of Minimal Pulmonary Tuberculosis," I. D. Bobrowitz, M.D., and Ralph E. Dwork, M.D.

"Why They Leave Against Advice," I. D. Bobrowitz, M.D.

"Recent Experiences in Canada and The United States," H. M. James, Clinical Tuberculosis Officer, Melbourne.

"The Value of Influenza Virus Baccine, Types A and B," Louis L. Friedman, M.D., Birmingham, Alabama.

"Treatment of Bronchial Asthma," Vincent J. Derbes, M.D., and Hugo T. Engelhardt, M.D., F.A.C.P., J. B. Lippincott Co., Philadelphia, June 1946.

"Promin Inhalation Therapy in Pulmonary Tuberculosis," James E. Edlin, M.D., I. D. Bobrowitz, M.D., F. K. Safford Jr., M.D., and Frank S. Butler, M.D.

"The Problems of Tuberculosis" — Reports on Tour of Canada and United States of America—(1) Administrative and Medical Section by Dr. H. M. James, (2) Surgical Aspects of Tuberculosis in U.S.A. and Canada in 1945 by Mr. C. J. Officer Brown.

"Medical Pioneers in the Near East," Dr. Halim J. Dewlett, Beirut, Labanon.